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RESPIRATORY CARE

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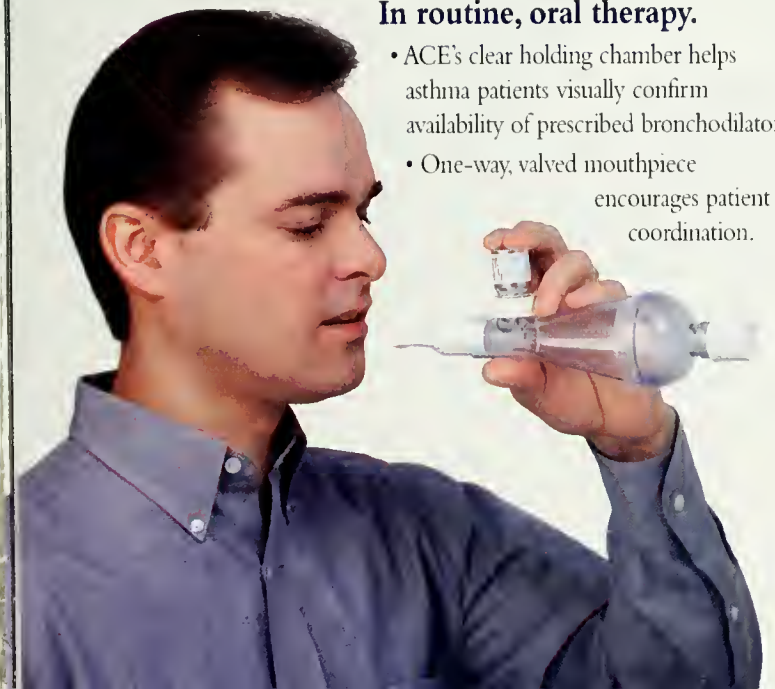

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1. Harwood, Robert, Rau, Joseph, Thomas-Goodfellow, Lynda. "A Comparison of Three Methods of Metered-Dose Bronchodilator Delivery to a Mechanically Ventilated Adult Lung Model." *Respiratory Care* September 1994 Vol. 39 No. 7 p. 696.
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Editorials, Commentaries, and Reviews To Note

Noninvasive Mechanical Ventilation in Patients with Acute Respiratory Failure (Review). N Abou-Shala, GU Meduri. Crit Care Med 1996;24(4)705-715.

Drug Therapy: Theophylline in Asthma (Review)—M Weinberger, L Hendeles. N Engl J Med 1996;334(21):1380.

Hospital-Acquired Pneumonia in Adults: Diagnosis, Assessment of Severity, Initial Antimicrobial Therapy, and Preventative Strategies (Consensus Statement)—American Thoracic Society. Am J Respir Crit Care Med 1995;153:1711-1725.

Persistent Pulmonary Hypertension of the Newborn and Smoking and Aspirin and Nonsteroidal Anti-inflammatory Drug Consumption during Pregnancy—LJ Van Marter, A Leviton, EN Allred, M Pagano, KF Sullivan, A Cohen, MF Epstein. Pediatrics 1996;97(5):658.

OBJECTIVE: Prenatal causation of persistent pulmonary hypertension of the newborn (PPHN) is suggested by a specific pattern of pulmonary vascular remodeling observed immediately after birth in some infants with fatal PPHN. The goal of this study was to determine whether PPHN is associated with fetal exposure to: (1) tobacco and marijuana smoking (ie, contributors to fetal hypoxemia), (2) consumption of aspirin and other nonsteroidal anti-inflammatory drugs (ie, inhibitors of prostaglandin synthesis), and (3) cocaine use (ie, a contributor to vasospasm). DESIGN: Case-control interview study. SETTING: Two Harvard-affiliated newborn intensive care units. PARTICIPANTS: Mothers of case infants who had PPHN or who met criteria for the referent group. INTERVENTIONS: During July 1985 through April 1989, we interviewed mothers of 103 infants with PPHN and 298 control infants. Because of potential selection bias that might result from recruiting only inborn control infants even though two-thirds of cases were outborn, separate analyses compared the 103 total and 35 inborn infants with PPHN with the 298 inborn control infants. Multivariate analyses were used to adjust for potential confounding factors, including maternal education and Medicaid health insurance (ie, 2 markers of socioeconomic status), other antenatal factors found to be associated with PPHN (ie, maternal urinary tract infection and diabetes mellitus), and the infant's sex. MAIN OUTCOME MEASURES: Self-reported use or consumption of tobacco, marijuana, cocaine, aspirin, and other nonsteroidal anti-inflammatory drugs during pregnancy. RESULTS: The adjusted odds ratios (and 95% confidence intervals) for maternal pregnancy exposures to the factors of principal interest among the total study population were: aspirin, 4.9 (1.6-15.3); and nonsteroidal anti-inflammatory drugs, 6.2 (1.8-21.8); for the inborn group they were: aspirin, 9.6 (2.4-39.0); and nonsteroidal anti-inflammatory drugs, 17.5 (4.3-71.6). Although the association between tobacco smoking during pregnancy and PPHN was elevated in univariate analyses, with odds ratios (and 95% confidence intervals) of 2.0 (1.2-3.4) and 1.3 (0.6-3.3) for total and inborn populations, respectively, the relationship was not significant after adjustment for all other factors in the final logistic regression model. Acknowledged illicit drug use was too infrequent (3.2%) to evaluate. CONCLUSION: Maternal consumption of nonsteroidal anti-inflammatory drugs and aspirin during pregnancy or the reasons these drugs were ingested seem to contribute to an increased risk of PPHN.

Antenatal Phenobarbital Therapy and Neonatal Outcome II: Neurodevelopmental Outcome at 36 Months—S Shankaran, E Woldt, J Nelson, M Bedard, V Delaney-Black. Pediatrics 1996;97(5):649.

OBJECTIVE: To evaluate the effect of antenatal phenobarbital (PB) therapy on neurodevelopmental outcome at 36 months. DESIGN: Prospective, randomized, controlled trial. SETTING: Single-institution study. SUBJECT & INTERVENTIONS: Children born to women who participated in the study evaluating the effect of antenatal phenobarbital (PB) on neonatal intracranial hemorrhage were prospectively followed to 3 years of age. OUTCOME MEASURES: Physical growth, neurologic examinations, and developmental testing (McCarthy Scales of Children's Abilities). Comparisons between groups were made on all children and those born to multiple gestations. RESULTS: Forty-one children born to women who received 10 mg/kg PB before delivery and 55 children in the control group were evaluated. Three children, all in the control group, had growth parameters (height, weight, and head circumference) below the fifth percentile. The McCarthy General Cognitive Index (standard, 100 ± 16) was 93 ± 20 in the PB group and 85 ± 18 in the control group. The subscores tended to be higher in the PB group than in the control group, with higher quantitative scores in the PB group (44 ± 11 vs 39 ± 8). Neurologic deficits were noted in 2 of 41 in the PB group and in 6 of 55 in the control group. CONCLUSIONS: Infants born to women who received antenatal PB therapy had similar neurodevelopmental outcomes as infants born to women who did not receive PB. No adverse effects of PB exposure were detected.

Influence of Referring Physicians on Interventions by a Pediatric and Neonatal Critical Care Transport Team—JB Kronick, TC Frewen, N Kissoon, R Lee, JF Sommerauer, WD Reid, S Casier, K Boyle. Pediatr Emerg Care 1996;12(2):73.

The objective of this study was to determine the influence of: (a) pediatrician versus nonpediatrician referrals on a transport team's therapeutic interventions and (b) referring physician's year of graduation on interventions performed by the transport team. From November 1987 through December 1989, we prospectively compared the therapeutic interventions performed by the critical care transport team on newborns and pediatric patients with the referring physician's specialty and year of graduation. The transport team (critical care physician [PL3 or greater], registered respiratory therapist, critical care nurse), recorded all therapeutic interventions, including both procedural and phar-

macologic, for 213 newborn and 149 consecutive pediatric transports. Referring physicians were categorized as pediatricians and nonpediatricians. Data were analyzed by analysis of variance, χ^2, or linear regression. All patients were admitted to either the pediatric or the neonatal intensive care unit, and over 80% of both age groups received assisted ventilation. Newborns referred by nonpediatricians required significantly more procedural interventions (2.64 vs 1.91, $p = 0.016$) than those referred by pediatricians. The opposite relationship was observed among pediatric patients in that children referred by pediatricians received more frequent intervention ($p = 0.008$) than those referred by nonpediatricians. There was a significant inverse relationship between the referring physician's year of medical school graduation and the number of therapeutic interventions (total interventions = 6.17 − 0.040 × graduation year, $p = 0.01$) and procedural interventions (procedural interventions = 3.54 − 0.024 × graduation year, $p = 0.01$). We found that the referring physicians' medical training affected the number of interventions their patients received. Similarly, patients were likely to receive more interventions if the referral physician's training was not recent. These data have educational implications and support the concepts of continuing medical education, recertification, and maintenance of skills among physicians providing care to critically ill newborns and pediatric patients.

Mechanical Ventilation and Arterial Blood Gas Measurements 24 Hours Postextracorporeal Life Support for Survivors of Pediatric Respiratory Failure—FW Moler, JM Palmisano, JR Custer, RH Bartlett. Crit Care Med 1996;24(4):679.

OBJECTIVE: To summarize our institutional experience concerning mechanical ventilation support and blood gas measurements in the 24-hour period following extracorporeal life support (ECLS) for pediatric acute respiratory failure. DESIGN: Descriptive study. SETTING: A tertiary pediatric referral center. PATIENTS: Children aged 1 month to 18 years treated with ECLS for acute respiratory failure at University of Michigan Hospitals from November 1982 to June 1993. All patients aged 1 month to 18 years who received ECLS for acute respiratory failure were included. Patients who received ECLS primarily for cardiac support or who had a diagnosis of congenital gastrointestinal malformation (ie, congenital diaphragmatic hernia) were excluded. INTERVENTIONS: ECLS for severe pediatric respiratory failure. MEASUREMENTS & MAIN RESULTS: Forty-nine children were treated at our center with ECLS for acute respiratory failure and 36 (73%) survived. Ventilator settings immediately after decannulation from ECLS for survivors were as follows: F_{IO_2} 0.53 ± 0.18 (SD); intermittent mandatory ventilation (IMV) 29.6 ± 11.8 breaths/min, positive end-expiratory pressure 5.3 ± 1.6 cm H_2O, mean airway pressure 12.6 ± 2.9 cm H_2O, and peak inspiratory pressure 31.7 ± 5.5 cm H_2O. Arterial blood gas measurements at decannulation were P_{aO_2} 89.4 ± 30.9 torr (11.9 ± 4.1 kPa), P_{aCO_2} 43.7 ± 9.7 torr (5.8 ± 1.3 kPa), and pH 7.39 ± 0.07. Twenty-four hours after decannulation, ventilator settings, and arterial blood gas measurements were as follows: F_{IO_2} 0.42 ± 0.14, IMV 27.4 ± 13.5 breaths/min, positive end-expiratory pressure 5.2 ± 1.6 cm H_2O, mean airway pressure 12.0 ± 3.4 cm H_2O, peak inspiratory pressure 31.1 ± 6.5 cm H_2O, P_{aO_2} 77.0 ± 16.9 torr (10.3 ± 2.3 kPa), P_{aCO_2} 44.9 ± 8.4 torr (6.0 ± 1.1 kPa), and pH 7.40 ± 0.07. Variables associated with oxygenation status ($P_{[A\text{-}a]O_2}$) and mean airway pressure (oxygenation index) improved during the immediate 24-hour period postbypass ($p < 0.05$). CONCLUSIONS: Successful decannulation from ECLS for >24 hours resulted in long-term survival in 97% (36/37) of children. Ventilator parameters and arterial blood gas measurements during the 24-hour period following bypass have been described for this population. Such conventional support may indicate safe levels of oxygen and mechanical ventilation pressures for the postbypass recovering lung.

Effect of Conventional Mechanical Ventilation and Jet Ventilation on Airway Pressure in Dogs and Plastic Models with Tracheal Stenosis—M Shinozaki, A Sueyoshi, T Morinaga, H Tsuda, T Muteki. Crit Care Med 1996;24(4):658.

OBJECTIVE: To evaluate the effect of jet ventilation on tracheal stenosis in dogs and plastic models. DESIGN: Prospective, randomized trial in dogs, and multitrial tests in tracheal stenosis models. SETTING: Animal laboratory in a university setting. INTERVENTIONS: Tracheal stenosis was surgically created around the middle of the trachea. Conventional mechanical ventilation and jet ventilation were compared at the same value of P_{aCO_2} in dogs and at the same tidal volume in tracheal stenosis models. SUBJECTS: Twelve mongrel dogs and 4 types of plastic models with combinations of short or long stenosis and fluid or nonfluid stenosis. MEASUREMENTS & MAIN RESULTS: Canine Studies—Mean peak airway pressure values at the distal and proximal portion of the stenosis, and the end-expiratory pressure at the distal portion of the stenosis, were significantly higher during conventional mechanical ventilation than during jet ventilation. The mean values of arterial pressure, pulmonary arterial pressure, central venous pressure, and cardiac output did not change significantly between conventional mechanical ventilation and jet ventilation, except for the pulmonary artery occlusion pressure value. Plastic Mold Studies—Peak airway pressure and end-expiratory airway pressure at the poststenotic trachea during jet ventilation with the model lung were significantly lower than during conventional mechanical ventilation. The difference in peak airway pressure and end-expiratory airway pressure values between jet ventilation and conventional mechanical ventilation increased more in short stenosis and nonfluid stenosis. CONCLUSIONS: The jet flow that struck the portion of the stenosed wall reversed direction, even during early expiration. Therefore, the expiration during jet ventilation was facilitated more by the reversed flow than by the expiration during conventional mechanical ventilation. This reversed flow may provide lower end-expiratory airway pressure at the poststenotic portion with jet ventilation than with conventional mechanical ventilation. We conclude that jet-ventilation was a useful method of ventilation in cases with tracheal stenosis, especially nonfluid and short stenosis.

Effect of Jet Ventilation on Heart Failure: Decreased Afterload but Negative Response in Left Ventricular End-Systolic Pressure-Volume Function—A Weber, M Mathru, MW Rooney. Crit Care Med 1996;24(4):647.

OBJECTIVES: To examine the mechanism of cardiac assist with systolic jet ventilation, specifically effects on loading conditions and left ventricular pressure-volume function. Both systolic and diastolic jet ventilation were compared in the absence and presence of heart failure. DESIGN:

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Prospective, 2-factor, repeated-measures study. SETTING: Animal laboratory. SUBJECTS: Ten anesthetized, closed-chest dogs. INTERVENTIONS: The measurement protocol consisted of 2 phases: (a) apnea, randomized jet ventilation (systole- and diastole-synchronized); (b) postjet ventilation apnea, before and after heart failure, induced with a propranolol-imipramine-plasma expansion treatment. MEASUREMENTS & MAIN RESULTS: Systolic or diastolic jet ventilation was associated with mean airway pressures of ≈7 mm Hg and intrapleural pressures of ≈3 mm Hg in both heart conditions. In normal hearts, jet ventilation (either mode) decreased transmural left ventricular end-diastolic pressure by 40% to 60% ($p < 0.05$), left ventricular end-diastolic volume 25 ± 8%, and stroke volume by 28% to 30%. Heart failure was associated with decreases (41 ± 6%) in end-systolic pressure-volume function (ie, pressure change/volume change or elastance), transmural left ventricular end-systolic pressure (22 ± 3%), and stroke volume (16 ± 4%), and increased transmural left ventricular end-diastolic pressure (139 ± 6%). Application of jet ventilation (either mode) during heart failure did not affect stroke volume but significantly ($p < 0.05$) attenuated transmural left ventricular end-diastolic pressure by 30% to 40%, left ventricular end-diastolic volumes by 33 ± 9%, and transmural left ventricular end-systolic pressure by 11% to 19% ($p < 0.05$). After jet ventilation, left ventricular elastance was decreased 36 ± 8% in normal hearts and 35 ± 11% in failing hearts. Stroke volume, however, returned to baseline levels because of increases in transmural left ventricular end-diastolic pressure in both heart conditions, and also in failing hearts, because transmural left ventricular end-systolic pressure remained decreased ≈30% ($p < 0.05$). CONCLUSIONS: Jet ventilation did not decrease stroke volume in failing hearts because of the afterload-reducing benefit (decreased transmural left ventricular end-systolic pressure) of increased intrapleural pressure in dilated ventricles. Moreover, jet ventilation did not have positive effects on myocardial function and had negative effects on left ventricular elastance in the postjet ventilation period in both normal and failing hearts. Cardiac assist by jet ventilation was not cycle specific, suggesting no selective benefit of jet ventilation over conventional positive-pressure ventilation during heart failure. These studies demonstrate a negative inotropy associated with jet ventilation that, during heart failure, may compromise the general benefit of positive-pressure-mediated increases in intrapleural pressure.

Modulating Effects of Propofol on Metabolic and Cardiopulmonary Responses to Stressful Intensive Care Unit Procedures—D Cohen, K Horiuchi, M Kemper, C Weissman. Crit Care Med 1996;24(4):612.

OBJECTIVE: Patients in the intensive care unit (ICU) undergo acute increases in metabolic and cardiopulmonary demands in response to routine care interventions, such as chest physical therapy. This study examined whether the short-acting drug, propofol, could blunt the responses to chest physical therapy. DESIGN: Prospective, randomized, crossover (placebo vs drug) study. SETTING: University hospital surgical ICU. PATIENTS: Postoperative ICU patients being ventilated in the synchronized intermittent mandatory ventilation mode. INTERVENTIONS: Two groups of 16 patients were studied. Each patient received 2 successive sessions of chest physical therapy. In random fashion, one was preceded by the administration of placebo and the other by an intravenous bolus of propofol (0.75 mg/kg in 1 group and 0.35 mg/kg in the other group). Each session was preceded and followed by a period of rest. MEASUREMENTS & MAIN RESULTS: The increases in oxygen uptake, CO_2 elimination, oxygen delivery, heart rate, and systolic blood pressure associated with chest physical therapy were attenuated with the low dose and suppressed with the high dose of propofol. The P_{aCO_2} concentration was slightly increased during both placebo and drug administration. CONCLUSIONS: Propofol, in the doses administered in this study, significantly reduced the hemodynamic and metabolic stresses caused by chest physical therapy.

Nonrespiratory Predictor of Mechanical Ventilation Dependency in Intensive Care Unit Patients—MJA Sapijaszko, R Brant, D Sandham, Y Berthiaume. Crit Care Med 1996;24(4):601.

OBJECTIVE: To determine the role of serum albumin concentration as a predictor of mechanical ventilation dependency. DESIGN: Prospective, observational trial. SETTING: Multidisciplinary intensive care unit (ICU) in a university hospital. PATIENTS: One hundred forty-five consecutive patients who required mechanical ventilation for >72 hours. INTERVENTIONS: Patients were classified into 5 different groups based on the cause of respiratory failure. The following parameters were recorded daily: serum albumin concentration; Acute Physiology and Chronic Health Evaluation II (APACHE II) score; and fluid balance. Using multiple regression, multiple logistic regression analysis, and the Anderson-Gill proportional hazards model, we determined the metabolic factors that could help predict weaning success. MEASUREMENTS & MAIN RESULTS: The mean length of ICU stay was 12.3 ± 1.0 days. The duration of mechanical ventilation dependency was 10.5 ± 1.0 days. The initial mean serum albumin concentration was 25.2 ± 0.6 g/L. The APACHE II score on the first day of ICU stay was 19.1 ± 0.6. Although albumin concentration was significantly lower and the APACHE II score was significantly higher in ICU nonsurvivors than in ICU survivors, albumin concentration on ICU admission was not a predictor of the length of time spent receiving mechanical ventilation. The profile of albumin concentration changes was different between weaned and

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The South Carolina Society for Respiratory Care
and the
American Association for Respiratory Care
present a

Special Pre-Convention Seminar

Monday, September 23, 1996 • Charleston, South Carolina

The 1996 Annual Convention of the South Carolina Society will be held Tuesday through Thursday, September 24-26, at the Hilton Hotel in beautiful Charleston. This year's convention will be preceded by a special seminar organized in cooperation with the American Association for Respiratory Care. The seminar will present timely information on how respiratory care practitioners and their employers can change and benefit from today's hospital restructuring programs. The seminar is approved for 6 hours of continuing respiratory care education (CRCE) credit by the AARC.

Successful Strategies for Tomorrow's Respiratory Care Services

9:00 a.m. - 9:05 a.m.
Program Overview

9:05 a.m. - 9:55 a.m.
The Needs of Health Care Systems in the Future—Emerging Trends and Expanding Skills Inventory presents the forces impacting on health care delivery and the response by delivery systems. Reviews past, present, and future reimbursement mechanisms and the response of delivery systems to incentives they create. Explains the impact of expanding post acute services, opportunities for RCPs, and the skill sets required to add value in future health care systems.

10:00 a.m. - 10:55 a.m.
Providing Value in Acute Care Facilities—Managing Demand for Services defines the value added by RCPs in present and future acute care facilities. Describes how to sell decision-makers on the value of using guidelines and protocols to ensure appropriate utilization. Defines resources and support required to implement a utilization control program. Presents a case study of a department that has been successful in ensuring appropriate utilization.

11:00 a.m. - 11:55 a.m.
The Post Acute Environment—Skilled Nursing Facilities, Subacute, and Home Care identifies market forces and governmental regulations that foster growth in the post acute environment. Discusses the opportunities for RCPs created by government and private managed care contracting. Reviews the mechanism for reimbursement of respiratory care services in post acute sites and the current HCFA scrutiny of transfer agreements.

1:30 p.m. - 2:25 p.m.
Respiratory Care in Group Practice and Disease Management explores opportunities for RCPs as mid-level providers in capitated physician group practice. Defines disease management and explains why asthma patients are ideal candidates for disease management programs. Identifies skill sets required by mid-level providers and explains why RCPs add unique value to respiratory disease management programs.

2:30 p.m. - 3:25 p.m.
Respiratory Care in the Future: Integrated Care Delivery Systems describes market forces encouraging the development of integrated care delivery systems. Reviews current and projected penetration of managed care across the nation. Explains the components of an integrated delivery system and the competitive advantage afforded by the system. Presents a proposed respiratory care service structure to meet the needs of an integrated care delivery system.

3:30 p.m. - 4:25 p.m.
Marketing and Selling Your Service and Your Services presents a systematic approach to developing new or expanded services. Discusses fundamentals of a business plan and techniques for successfully presenting ideas to organizational decision-makers. Reviews common pitfalls to avoid.

For additional program information, registration form, and hotel reservation for this special seminar and the South Carolina Society annual meeting, please

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mechanical ventilation-dependent patients. At the time of weaning patients from the ventilator, the median albumin concentration was higher than in those patients who continued to be supported by mechanical ventilation. This effect of albumin could not be attributed to patient fluid balance or to the severity of illness since each factor had an independent influence in predicting weaning, using the Anderson-Gill proportional hazards model. CONCLUSIONS: Initial serum albumin concentration did not necessarily predict weaning success. However, when serum albumin concentration was assessed on a daily basis, its trend was important in determining the relative chance of being successfully weaned from the ventilator. This finding suggests that albumin may be an index of the metabolic status of the patient, which could be important in determining the weanability of the patients who are mechanically ventilated for prolonged periods of time.

Adult Respiratory Distress Syndrome: A Systematic Overview of Incidence and Risk Factors—BG Garber, PC Hébert, JD Yelle, RV Hodder, J McGowan. Crit Care Med 1996;24(4):687.

OBJECTIVE: To determine the published incidence of adult respiratory distress syndrome (ARDS) as well as the clinical evidence supporting a causal association between ARDS and its major risk factors. DATA SOURCES: The National Library of Medicine MEDLINE database and the bibliographies of selected articles. STUDY SELECTION: Clinical studies were selected from the English literature, if they pertained to either the incidence of ARDS or its association with 1 or more commonly identified risk factors. DATA EXTRACTION: All relevant studies identified by the search were evaluated for strength of design, and risk factors were scored according to established criteria for the strength of causation. DATA SYNTHESIS: A total of 83 articles were considered relevant: 6 on incidence and 77 on risk factors. Only 49% of the 83 articles provided a definition of ARDS; a definition of risk factors was given in 64%, and 23% had no definition for either ARDS or risk factors. The published, population-based incidence of ARDS ranges from 1.5 to $5.3/10^5$ population/year. The strongest clinical evidence supporting a cause-effect relationship was identified for sepsis, aspiration, trauma, and multiple transfusions. The weakest clinical evidence was identified for disseminated intravascular coagulation. The following study types were represented by the 77 articles on risk factors: observational case-series (56%); cohorts (23%); case-controls (12%); nonrandomized clinical trials (5%); and randomized clinical trials (3%). Only a single study reported an odds ratio. CONCLUSIONS: The significant variation in the incidence of ARDS is attributed to differences in the type and strength of study designs, as well as definitions of ARDS. While a substantial body of evidence exists concerning a causal role for ARDS risk factors, such as sepsis, aspiration, and trauma, >60% of clinical studies employed weak designs. The lack of reproducible definitions for ARDS or its potential risk factors in 49% of studies raises concerns about the validity of the conclusions of these studies regarding the association between ARDS and the supposed risk factors. *See the related editorial: **Acute Respiratory Distress Syndrome: Consensus, Definitions, and Future Directions**. TL Petty. Crit Care Med 1996;24(4):555-556.*

Chest Physiotherapy and Postextubation Atelectasis in Infants—S Al-Alaiyan, D Dyer, B Khan. Pediatr Pulmonol 1996;21:227.

We investigated the role of chest physiotherapy (CPT) in preventing postextubation atelectasis (PEA) in infants. Sixty-three infants who were admitted to the neonatal intensive care unit and intubated for more than 24 hours and who showed no evidence of atelectasis by chest x-ray prior to extubation were enrolled in the study. Infants were randomly assigned to 2-hourly CPT, 4-hourly CPT, or a no CPT group. Chest physiotherapy began immediately after extubation and consisted of postural drainage, bilateral chest vibration, and suctioning. A second chest x-ray was obtained on all infants 24 hours following extubation. The 3 groups were comparable in birthweight, gestational age, and duration of intubation. In the 24-hour period following extubation, the incidence of PEA was not statistically significant in the 3 groups ($p = 0.33$). Two infants in the 2-hourly CPT group were placed on nasal continuous positive airway pressure; 2 in each of the 2-hourly and the no CPT groups required re-intubation and intermittent positive pressure ventilation to treat symptomatic atelectasis. We conclude that postextubation chest physiotherapy as used in this study did not prevent atelectasis in extubated infants.

Histopathologic and Microbiologic Aspects of Ventilator-Associated Pneumonia—N Fàbregas, A Torres, M El-Ebiary, J Ramírez, C Hernández, J González, J Puig de la Bellacasa, MT Jiménez de Anta, R Rodriguez-Roisin. Anesthesiology 1996;84(4):760.

BACKGROUND: The relationship between microbiology and histology in patients with ventilator-associated pneumonia has been sparsely described. METHODS: Twenty-five patients who died in the intensive care unit after their lungs had been mechanically ventilated for 72 hours were studied. Twenty of the 25 died with clinical suspicion of pulmonary infection. A total of 375 immediate postmortem pulmonary biopsies were obtained after death and processed for quantitative microbiology and histology. Four evolutionary stages of pneumonia were defined: early, intermediate, advanced, and resolution. RESULTS: At least 1 specimen with histologic evidence of pneumonia was found in all but 2 patients (92%). Histologic pneumonia was a widespread and frequent process (46% of biopsies examined) involving predominantly the lower lobes (55% of all biopsies with pneumonia) and showing different histopathologic stages of progression coexisting in the same lung lobes. Lung cultures were frequently polymicrobial (149 of 375, 40% of the pulmonary biopsy cultures, and 20 of 25, 80% of the cases) and not always yielding the same pathogen (19 microorganisms) when comparing 1 lung to the other. Histopathology and microbiologic biopsy cultures showed a weak relationship (28% and 49% of species had counts $\geq 10^3$ cfu/g in samples without pneumonia from patients with and without prior antibiotic treatment, respectively). Histopathologic evolutionary stages were not associated with any differences in quantitative culture results of pulmonary biopsies, independently of prior administration of antibiotics. Higher bacterial concentrations of biopsy cultures were associated with the absence of prior antibiotic treatment. CONCLUSIONS: Ventilator-associated pneumonia is a frequent diffuse and polymicrobial process showing different coexisting degrees of evolution and involving preferentially the lower lobes. Microbiology and histology can be dissociated even in the absence of prior antibiotic treatment. Lung histology appears more reliable than bacteriology as a diagnostic reference test.

Predictors of Pulse Oximetry Data Failure—DL Reich, A Timcenko, CA Bodian, J Kraidin, J Hofman, M DePerio, SN Konstadt, T Kurki, JB Eisenkraft. Anesthesiology, 1996;84(4):859.

BACKGROUND: Pulse oximeters have been reported to fail to record data in 1.12-2.50% of cases in which anesthesia records were handwritten. There is reason to believe that these may be underestimates. Computerized anesthesia records may provide insight into the true incidence of pulse oximetry data failures and factors that are associated with such failures. METHODS: The current study reviewed case files of 9,203 computerized anesthesia records. Pulse oximetry data failure was defined as the presence of at least 1 continuous gap in data ≥10 minutes in duration in a case. A multivariate logistic regression model was used to identify predictors of pulse oximetry data failure, and a modified case-control method was used to determine whether extremes of blood pressure and hypothermia during the procedure were associated with pulse oximetry data failure. RESULTS: The overall incidence of cases that had at least 1 continuous gap of ≥10 minutes in pulse oximetry data was 9.18%. The independent preoperative predictors of pulse oximetry data failure were ASA physical status 3, 4, or 5 and orthopedic, vascular, and cardiac surgery. Intraoperative hypothermia, hypotension, hypertension, and duration of procedure were also independent risk factors for pulse oximetry data failure. CONCLUSIONS: Pulse oximetry data failure rates based on review of computerized records were markedly greater than those previously

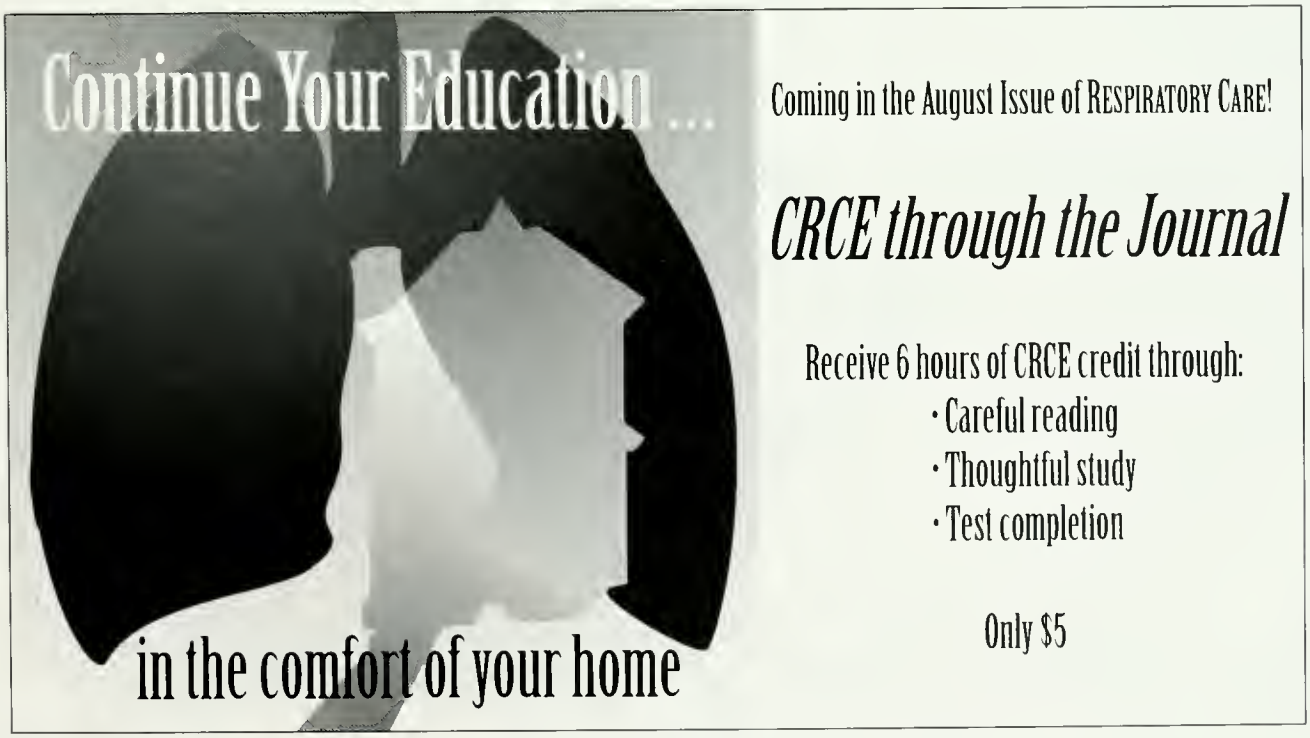
reported. Physical status, type of surgery, and intraoperative variables were risk factors for pulse oximetry data failure. Regulations and expectations regarding pulse oximetry monitoring should reflect the limitations of the technology.

Ventilation with Constant versus Decelerating Inspiratory Flow in Experimentally Induced Acute Respiratory Failure—AM Markström, M Lichtwarck-Aschoff, BA Svensson, KA Nordgren, UH Sjöstrand. Anesthesiology 1996;84 (4):882.

BACKGROUND: Recognition of the potential for ventilator-associated lung injury has renewed the debate on the importance of the inspiratory flow pattern. The aim of this study was to determine whether a ventilatory pattern with decelerating inspiratory flow, with the major part of the tidal volume delivered early, would increase functional residual capacity at unchanged (or even reduced) inspiratory airway pressures and improve gas exchange at different positive end-expiratory pressure levels. METHODS: Surfactant depletion was induced by repeated bronchoalveolar lavage in 13 anesthetized piglets. Decelerating and constant inspiratory flow ventilation was applied at positive end-expiratory pressure levels of 22, 17, 13, 9, and 4 cm H_2O. Tidal volume, inspiration-to-expiration ratio, and ventilatory frequency were kept constant. Airway pressures, gas exchange, functional residual capacity (using a wash-in/ washout method with sulfurhexafluoride), central hemodynamics, and extravascular lung water (using the thermo-dye-indicator dilution technique) were measured. RESULTS: Decelerating inspiratory flow yielded a lower arterial carbon dioxide tension compared to constant flow, that is, it improved alveolar ventilation. There were no differences between the flow patterns regarding end-inspiratory occlusion airway pressure, end-inspiratory lung volume, static compliance, or arterial oxygen tension. No differences were seen in hemodynamics and oxygen delivery. CONCLUSIONS: The decelerating inspiratory flow pattern increased carbon dioxide elimination, without any reduction of inspiratory airway pressure or apparent improvement in arterial oxygen tension. It remains to be established whether these differences are sufficiently pronounced to justify therapeutic consideration.

Short-Term Compliance with Peak Flow Monitoring: Results from a Study of Inner City Children with Asthma—S Redline, EC Wright, M Kattan, C Keresmar, K Weiss. Pediatr Pulmonol 1996;21:203.

The objective of the study was to assess the feasibility of initiating daily peak flow monitoring in a research study of asthma in inner city children. We performed a descriptive study of patterns of peak flow monitoring in children randomized to receive a simple mini-Wright (SM) or an electronic recording meter (ERM). The ERM served as a "covert" meter, providing objective documentation of actual peak flow use. Sixty-five Hispanic or African-American children, ages 5-9 years, with a history of physician-diagnosed asthma participated in the study. All children resided in census tracts with 40% or more of the population living at or below the poverty level. Subjects were instructed to use a peak flow meter (the SM or ERM) at least twice daily over a 3-week period, and to record peak flow values in a paper diary. Subjects who received the ERM were not made aware that measurements were also recorded electronically. Differences in patterns of use of the SM and ERM were assessed with the Wilcoxon signed rank test and Wilcoxon sum rank test. Adherence to peak flow monitoring was evaluated by comparing the percent days with missing values in the manually completed diary with those obtained by computer record. The Friedman statistic was used to compare changes in compliance (percent of days with missing peak flow entries) over time. Accuracy of peak flow readings was assessed by comparing the manual and electronic recordings with paired and unpaired *t*-tests and with Pearson product moment correlations. The percent of days with missing peak flow entries on diaries increased from 1.4% to 10.6% from the first to third week of monitoring ($p < 0.004$). The ERMs indicated a significantly greater percent of missing data than did the manual records ($p < 0.0002$). The difference in the percent of missing data for the electronic and manual records was most notable during the third study week, when the ERM and the manually completed records indicated that 52% and 15% of days, respectively, were without peak flow measures. Large inter-subject variations in the relationship between manually and electronically recorded peak flow measurements were observed, suggesting that errors in reading and transcribing peak flow rates occur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in a subset of asthmatics. We conclude that children and caretakers in the inner city may have considerable difficulty initiating and maintaining peak flow recordings. Data obtained by manual records may considerably overestimate actual use. Compliance with monitoring decreases markedly between the first and third week of monitoring.

Theophylline in Acute Childhood Asthma: A Meta-Analysis of Its Efficacy—DC Goodman, B Littenberg, GT O'Connor, JG Brooks. Pediatr Pulmonol 1996;21:211.

Although theophylline is a widely used drug for the treatment of acute childhood asthma, its efficacy has not been clearly established. This study constitutes a meta-analysis of published randomized clinical trials of theophylline in children hospitalized with acute asthma. We conducted a search of English language MEDLINE citations from 1966 to 1995 and analyzed the methods of each report meeting study criteria. We pooled similar clinical measures across studies if a test for homogeneity of effect size was nonsignificant. The 6 methodologically acceptable randomized clinical trials included a total of 164 children less than 18 years of age. Incomplete reporting of measures and variances was common. No study included children in intensive care settings. Using pooled results, pulmonary function parameters [forced expired volume in 1 second (FEV_1), forced expired flow (FEF)] appeared better at 24 hours in the theophylline group, but the results did not reach statistical significance (mean effect difference, +3.9% predicted values; pooled effect size, + 1.6 SDS; $p = 0.25$). A mean of 2.1 more albuterol treatments were administered in the theophylline group (pooled effect size, –0.18 SDS; $p = 0.02$), and the mean hospital stay was slightly longer (mean effect difference, –0.31 days; pooled effect size, –0.18 SDS; $p = 0.03$). We conclude that currently available data do not indicate a significant beneficial effect of theophylline in children hospitalized with acute asthma. There is evidence for weak detrimental effects. Theophylline efficacy in intensive care unit settings remains unstudied.

Effectiveness of Budesonide Aerosol in Ventilator-Dependent Preterm Babies: A Preliminary Report—S Arnon, J Grigg, M Silverman. Pediatr Pulmonol 1996;21:231.

The aim of this randomized, double-blind, placebo-controlled trial was to assess the short-term effect of a topical glucocorticoid (budesonide 600 μg twice daily) versus placebo administered by metered dose inhaler (MDI) and spacer (Aerochamber MV15) directly into the endotracheal tube of intubated infants for 7 days. Twenty preterm infants (mean birthweight, 1,030 g; mean gestational age, 27.3 weeks) who still needed assisted ventilation at 14 days of age were randomly assigned to receive budesonide ($n = 9$) or placebo ($n = 11$) and completed the study. The primary outcome was the need for mechanical ventilation after 7 days of treatment. Other outcome variables included ventilator settings, blood gases, serum cortisol levels, and bronchoalveolar lavage inflammatory cell counts. No ventilated infant was extubated during the study period. The treatment group showed significant improvements in mean peak inspiratory pressure, ventilator efficiency index, and (A-a) oxygen difference. There were no changes in the placebo group. Serum cortisol levels and bronchoalveolar lavage cell counts did not change significantly during the study period. There was no difference in side effects between the groups. This trial demonstrates that topical budesonide administered by MDI and Aerochamber produces clinical improvement in ventilated preterm infants, without glucocorticoid side effects.

Efforts To Reduce the Toll of Injuries in Childhood Require Expanded Research—Committee on Injury and Poison Prevention. Pediatrics 1996; 97(5):765.

Efforts to control injury, like those to control disease, are based on epidemiologic evidence that most occurrences are both predictable and preventable. Effective injury control programs depend heavily on evidence generated as a result of a strong research agenda. This policy statement outlines the magnitude of the injury toll and the prevention efforts that have expanded during the past

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The West Virginia Society for Respiratory Care
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Special Pre-Convention Seminar

Monday, September 30, 1996 • Huntington, West Virginia

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For additional program information, registration form, and hotel reservation for this special seminar and the West Virginia Society annual meeting, please

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Successful Strategies for Tomorrow's Respiratory Care Services

10:00 a.m. - 10:05 a.m.
Program Overview

10:05 a.m. - 10:50 a.m.
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11:45 a.m. - 12:30 p.m.
The Post Acute Environment—Skilled Nursing Facilities, Subacute, and Home Care
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Marketing and Selling Your Service and Your Services
Presents a systematic approach to developing new or expanded services. Discusses fundamentals of a business plan and techniques for successfully presenting ideas to organizational decision-makers. Reviews common pitfalls to avoid.

decade. Even with this expansion, the level of federal commitment to research aimed at reducing the toll of injury does not match the magnitude of the injury problem. Much research remains to be done, and the AAP recommends a multilevel and multifaceted approach to prevention and treatment, based on injury morbidity and mortality data. As injury control methods of proven efficacy continue to be developed, pediatricians can apply them to their patients, and thereby reduce the toll of childhood injury.

Muscle Weakness in Mechanically Ventilated Patients with Severe Asthma—JW Leatherman, WL Fluegel, WS David, SF Davies, C Iber. Am J Respir Crit Care Med 1996;153:1686.

Patients who undergo mechanical ventilation for severe asthma are at risk of developing diffuse muscle weakness because of acute myopathy. The relative importance of corticosteroids and neuromuscular paralysis in causing the myopathy is controversial, and it is uncertain whether the chemical structure of the drug used to induce paralysis influences the risk of myopathy. Using a retrospective cohort study design, we evaluated 107 consecutive episodes of mechanical ventilation for severe asthma to assess (1) the incidence of clinically significant weakness in patients treated with corticosteroids alone versus corticosteroids with neuromuscular paralysis, (2) the influence of the duration of paralysis on the incidence of muscle weakness, and (3) the relative risk of weakness in patients paralyzed with the nonsteroidal drug atracurium versus an aminosteroid paralytic agent (pancuronium, vecuronium). The use of corticosteroids and a neuromuscular blocking agent was associated with a much higher incidence of muscle weakness as compared with the use of corticosteroids alone (20 of 69 vs 0 of 38, $p < 0.001$). The 20 weak patients were paralyzed significantly longer than the 49 patients who received a neuromuscular blocking agent without subsequent weakness (3.4 ± 2.4 vs 0.6 ± 0.7 days, $p < 0.001$). Eighteen of the 20 weak patients had been paralyzed for more than 24 hours. The incidence of weakness was not reduced when paralysis was achieved with atracurium as opposed to an aminosteroid neuromuscular blocking agent. In conclusion, corticosteroid-treated patients with severe asthma who undergo prolonged neuromuscular paralysis are at significant risk for the development of muscle weakness, and the risk of weakness is not reduced by use of atracurium.

Efficacy of Dead-Space Washout in Mechanically Ventilated Premature Newborns—C Danan, G Dassieu, JC Janaud, L Brochard. Am J Respir Crit Care Med 1996;153:1571.

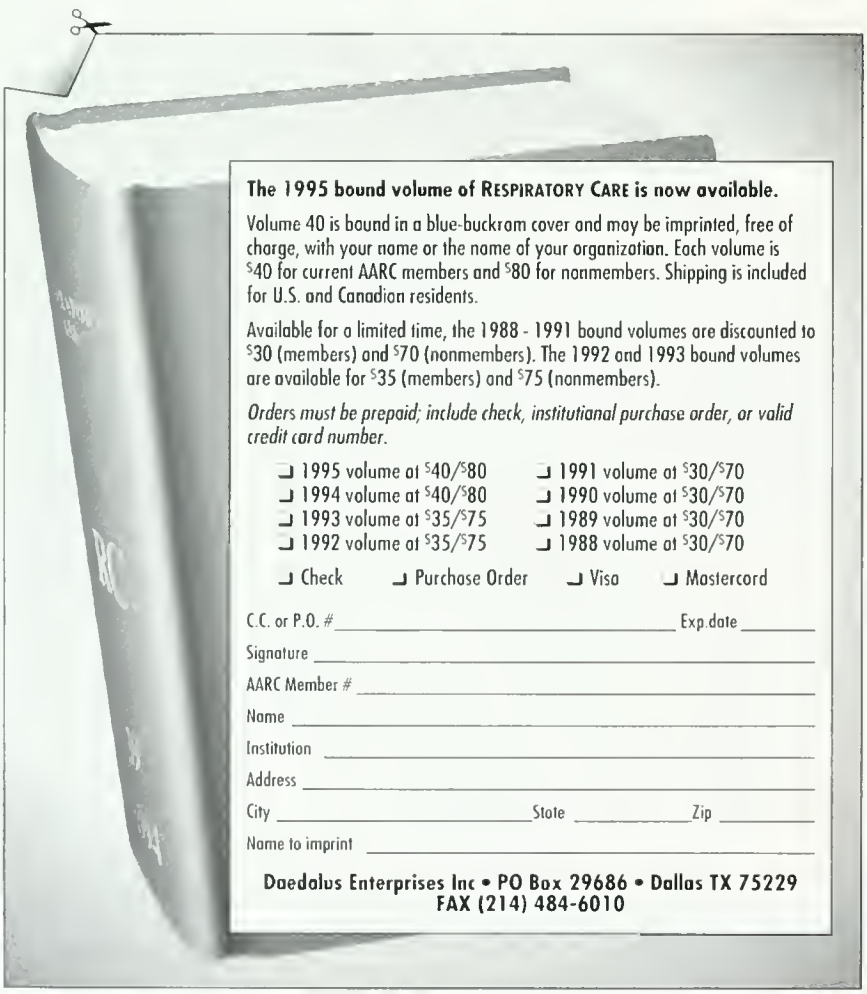
The prosthetic dead space makes a significant contribution to the total dead space in low-birthweight premature newborns receiving artificial ventilation in response to respiratory distress. Use of an endotracheal tube with capillaries molded into the tube wall enables washout of the dead space without insertion of a tracheal catheter. In 10 premature newborns (mean gestational age, 27.5 ± 2.2 weeks; mean weight, 890 ± 260 g) receiving continuous positive-pressure ventilation ($\bar{P}_{aw}$ = 12.7 ± 1.8 cm H_2O; F_{IO_2} = 39 ± 17%), tracheal gas insufflation (TGI) for CO_2 washout was conducted using this technique. The flow for tracheal insufflation (0.5 L/min) was derived from the inspiratory line of the ventilator circuit and blown into the trachea. Intratracheal pressures showed little or no TGI-related modification (< 1 cm H_2O). A control system enabled TGI discontinuation in the event of a pressure rise. At constant ventilation pressure, P_{aCO_2} decreased by 12.1 ± 5.9 mm Hg (ΔP_{aCO_2} = −26 ± 12%) under TGI, whereas P_{aO_2} remained unchanged. While maintaining P_{aCO_2} constant, peak inspiratory pressure (PIP) was decreased by 5.4 ± 1.7 cm H_2O ($\Delta PIP = -22.0 \pm 8.3\%$). TGI showed immediate efficacy (P_{CO_2} reduction of at least 5 mm Hg) in 9 of the 10 newborns who then received chronic TGI (14 to 138 hours). TGI appears to be an effective method, suitable for long-term clinical application, enabling a reduction in the aggressive nature of conventional ventilation.

Safe Transportation of Premature and Low Birthweight Infants—Committee on Injury and Poison Prevention and Committee on Fetus and Newborn. Pediatrics 1996;97(5):758.

Special considerations are essential to ensure the safe transportation of premature and low birthweight infants. Both physical and physiologic issues must be considered in the proper positioning of these infants. This statement discusses current recommendations based on the latest research and provides guidelines for physicians who counsel parents of very small infants on the choice of the best car safety seats for their infants.

Nebulized Budesonide Is as Effective as Nebulized Adrenaline in Moderately Severe Croup—D Fitzgerald, C Mellis, M Johnson, H Allen, P Cooper, P Van Asperen. Pediatrics 1996; 97(5):722.

OBJECTIVE: Nebulized budesonide and nebulized adrenaline have been shown to be effective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in the treatment of moderately severe croup. However, there has been no direct comparison of these therapies. We undertook a multicenter, randomized, double-blind, parallel group study in 66 hospitalized children with viral or spasmodic croup. METHODS: Children 0.5 to 6 years of age were assessed using a validated croup symptom score (stridor, 0 through 4; cough, 0 through 3; retractions, 0 through 3; dyspnea, 0 through 3; and color, 0 through 4) at 0.5, 1, 1.5, 2, 12, and 24 hours after nebulization. Patients received either budesonide (2 mg/4 mL) or L-adrenaline (4 mg/4 mL) via nebulization. The primary outcome measure was change in the total croup symptom score. RESULTS: Thirty-five children received budesonide and 31 received adrenaline. There was no significant difference in baseline features, including croup score (mean [95% confidence interval]: budesonide, 7.1 [6.7 – 7.5]; adrenaline, 7.7 [7.3 – 8.1]). All patients had significant improvement from baseline, and there was no significant difference between the 2 treatments, as measured by change in croup scores, change in oxygen saturation, duration of hospitalization, number of subsequent treatments with systemic steroids or adrenaline, and adverse events. No child required intubation. CONCLUSION: This study does not show any difference in efficacy and safety between nebulized budesonide and nebulized adrenaline in the treatment of acute upper airway obstruction in patients with moderately severe croup.

Pertussis Deaths: Report of 23 Cases in the United States, 1992 and 1993—N Wortis, PM Strebel, M Wharton, B Bardenheier, IRB Hardy. Pediatrics 1996;97(5):607.

OBJECTIVE: To characterize pertussis deaths and to identify possible risk factors and prevention strategies. METHODS: A retrospective review of all deaths attributed to pertussis with disease onset during 1992 and 1993 reported to the Centers for Disease Control and Prevention. Hospital discharge summaries and autopsy reports were reviewed, and additional clinical information was provided by physicians involved in the care of the children. RESULTS: During 1992 and 1993, 23 deaths attributed to pertussis were reported to the Centers for Disease Control and Prevention. Cultures for *Bordetella pertussis* were positive in 18 (90%) of the 20 cases in which it was performed. Twenty (87%) of the 23 children who died were younger than 1 year of age, and 18 (78%) of the children had received no doses of pertussis vaccine. Among 20 children for whom gestational ages were known, 12 (60%) were born at 36 weeks' gestation or earlier; in contrast, 10.7% of live births in the United States in 1992 were at 36 weeks' gestation or earlier. The median age of mothers whose children had fatal pertussis was 20 (range, 14 to 37) years in the 15 cases in which ages were known, compared with the national median age of 26.3 years in 1992. Pneumonia was a complication in all but 1 (96%) of the cases. Seizures occurred in 4 cases (17%), and acute encephalopathy occurred in 3 cases (13%). CONCLUSIONS: Pertussis continues to cause serious illness and death in the United States, particularly among infants who are not vaccinated. Preterm delivery and young maternal age may place infants at increased risk of death because of pertussis. Under the current pertussis vaccination schedule, three fourths of the infants who died were too young to have received 3 doses of pertussis vaccine, the minimum number of doses considered necessary for adequate protection against clinical pertussis. Additional strategies to prevent deaths caused by pertussis in young infants, such as starting infant vaccination at an earlier age and booster doses to adolescents and adults, need to be evaluated.

Birthweight-Specific Mortality for Extremely Low Birthweight Infants Vanishes by Four Days of Life: Epidemiology and Ethics in the Neonatal Intensive Care Unit—W Meadow, T Reimshisel, J Lantos. Pediatrics 1996;97(5):636.

BACKGROUND: The persistent differences between those who question the appropriateness of aggressive resuscitative measures for many extremely low birthweight (ELBW) infants (birthweight <1,001 g) and those who generally initiate such treatment has been a source of ongoing tension for physicians, parents, judges, and policy makers. We believe that much of this tension may be a result of the way the issue is framed. We began this study with the intuition that although many ELBW infants die, most succumb quickly. Were this true, discussions that considered only survival rates might miss the point. A more relevant statistic might be the degree to which interventions prolong dying, extend suffering, or use resources for infants who will eventually die. METHODS: We determined the survival and nonsurvival for 429 ELBW infants admitted to our neonatal intensive care unit (NICU) for 3 years. We noted particularly the relationship between birthweight, illness severity (fraction of inspired oxygen [F_{IO_2}] requirement for infants requiring mechanical ventilation), and the time course of mortality for nonsurvivors. We next calculated a resource utilization index (NICU bed days occupied by survivors and nonsurvivors) for each patient and for the population as a whole. Finally, we determined how NICU resources were distributed among infants who eventually died and those who survived. RESULTS: Of the 429 ELBW infants alive on day of life (DOL) 1,202 (47%) survived to be discharged. On DOL 1, both birthweight and illness severity independently predicted likelihood of survival. Approximately 80% of ELBW deaths occurred in the first 3 days of life—consequently, once an infant had survived to DOL 4, the likelihood of survival was dramatically enhanced (81% for the 249 patients alive on DOL 4). In addition, although survival for DOL 4 infants continued to depend on illness severity, survival no longer depended on birthweight. These observations on DOL 4 were confirmed in the subpopulation of 212 infants whose birthweight was <750 g. Overall, although 53% of ELBW babies admitted died, only ≈13% of all NICU bed-days (a proxy for resource allocation) were devoted to infants who did not survive. This figure did not vary as a function of birthweight. CONCLUSIONS: Generally, when we talk of survival rates to parents, ethics committees, or policy makers, we base our predictions largely on birthweight. The data presented here suggest that predictions should be corrected by including DOL and that, when this is done, the prognostic value of birthweight rapidly diminishes. In addition, birthweight-specific mortality and day of death for nonsurvivors correlated inversely; that is, more of the smaller infants died, but the doomed ones died more quickly. Consequently, medical resources allocated to nonsurvivors remained low, and independent of birthweight. This formulation lends weight both to the reasonableness of physicians in offering NICU care to ELBW infants with unlikely prospects for survival, and of parents and surrogate decision makers in requesting/ assenting to it.

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The Story of Mechanical Ventilator Circuits and Ventilator-Associated Pneumonia: A Model for Practice Changes in the Era of Managed Care

In this issue of RESPIRATORY CARE, Thompson describes the experience in a long-term ventilator unit in going from a practice of 7-day ventilator circuit changes to 14-day changes.[1] Using a before-after study design, this author demonstrated that the incidence of ventilator-associated pneumonia (VAP) was similar regardless of the time interval used for ventilator circuit changes (7-day circuit changes: 1.95 cases of VAP per 1,000 ventilator days; 14-day circuit changes: 1.63 cases of VAP per 1,000 ventilator days). Similar results were obtained in spontaneously breathing tracheostomy patients.

It is important to note that the design of this investigation has several important limitations that restrict the general application of the results. First, an historical control group was used, which did not adequately control for ongoing changes in practice over time at the study facility. Second, severity of illness between the 2 study groups was not compared. A number of standardized scoring systems currently exist, which can be used in clinical trials of critically ill patients for purposes of group comparisons.[2] Third, the clinical diagnosis of VAP employed did not rely on lower airway cultures obtained either bronchoscopically or nonbronchoscopically. However, similar clinical criteria for the diagnosis of VAP have been suggested to be adequate for the performance of high quality clinical research in other settings.[3] Fourth, only a small sample was examined, which limits the power of the study and increases the possibility of a type-II error (ie, wrongly accepting the null hypothesis, or no difference between the methods). However, despite these limitations, the author of this investigation is to be commended for his contribution because it targets a previously unstudied patient population. This investigation represents a much needed step in identifying the optimal frequency of ventilator circuit changes among long-term ventilator-dependent patients.[4]

The limitations noted require that additional outcome studies be performed in long-term ventilator-dependent patients. If these preliminary results can be confirmed in subsequent investigations, then stronger recommendations regarding the use of 14-day circuit changes—or possibly even no routine circuit changes—can be made. Qualities of evidence for grading the available medical literature in order to make specific recommendations regarding medical practices are published and have already been applied to patients requiring mechanical ventilation.[5,6] At present, the results of available randomized controlled trials and large before-after investigations strongly suggest that extended use of ventilator circuits is a safe practice among patients requiring mechanical ventilation in the acute care setting.[7-10] These studies have built upon earlier observational investigations, which suggested that less frequent ventilator circuit changes could be safe.[11,12] In fact, several studies have found that the incidence of VAP is actually increased among patients undergoing more frequent ventilator circuit changes.[7,12] These studies support the concept that it is not simply bacterial colonization of the ventilator circuit that predisposes to the development of VAP, but rather the aspiration of contaminated liquid boluses that may be more likely to occur with manipulation of the ventilator circuit.[10,13]

Based on the currently available experimental data, it is expected that the Centers for Disease Control and Prevention (CDC) will change their recommendations regarding the extended use of ventilator circuits among acutely ill patients in favor of less frequent ventilator circuit changes. In the 1994 Guideline, the CDC gave no clear recommendation for the maximum length of time that ventilator circuits can be safely left in place during prolonged periods of mechanical ventilation.[14] Although it appears that routine ventilator circuit changes are not required for patients receiving short-term mechanical ventilation (ie, < 14 days),[7-10] similar recommendations for long-term ventilator-dependent patients cannot be made at the present time. Only after additional rigorously performed studies have been conducted addressing this issue in this subgroup of ventilated patients can more specific recommendations be made.

Probably the most important aspect of the ventilator circuit story has been the stepwise systematic evaluation of this problem over a period of 2 decades. This has resulted in the development of cost-saving practice changes. In this current era of managed care, with its emphasis on medical cost reductions and patient outcomes, changes in medical practices have often occurred in order to reduce costs without first investigating their impact on patient outcomes. Intensive care units have frequently been targeted for cost reductions because they account for 10 to 15% of all hospital beds and for over 20% of all inpatient hospital costs, roughly equaling 1.4% of the gross domestic product.[15] The potential detrimental effects of such policies are illustrated by several recent studies demonstrating that the incidence of adverse patient

outcomes (eg, prolonged weaning times, iatrogenic events) may be increased as a result of decreased nursing services in the intensive care setting.[16,17] The ventilator circuit story demonstrates that cost-saving changes in medical practices that do not adversely impact upon individual patient outcomes can be identified. This experience should serve as the paradigm for conducting outcomes research and establishing future practice changes in the ICU setting.

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A SCoP Computer Model of Steady-State Gas Exchange: Evaluating the Oxygen-Tension-Based Indices of Venous Admixture

Wesley M Granger PhD RRT

BACKGROUND: The purpose of this computer model study was to develop a mathematical model of steady-state gas exchange and incorporate these equations in a computer simulation that could accurately predict the venous admixture, or physiologic shunt fraction ($\dot{Q}_s/\dot{Q}_t$), and P_{aO_2}. METHODS: A computer model was constructed to include interactions of O_2 and CO_2 dissociation curves and end-capillary, mixed-venous, and arterial blood gas values. Predictions of the computer model are used to assess the variability of the 5 most common indices of gas exchange—$P_{(A-a)O_2}$, P_{aO_2}/P_{AO_2}, P_{aO_2}/F_{IO_2}, estimated venous admixture (EVA), and Respiratory Index ($[P_{(A-a)O_2}]/P_{aO_2}$). The ability of the model to predict $\dot{Q}_s/\dot{Q}_t$ and P_{aO_2} was validated using bias and limits of agreement between model-generated data and traditional indices of gas exchange in 50 sets of data from 37 patients. RESULTS: The magnitude of the change in P_{aO_2}/P_{AO_2} is less than that of $P_{(A-a)O_2}$. However, for any given $\dot{Q}_s/\dot{Q}_t$ value, P_{aO_2}/P_{AO_2} varies widely. If $\dot{Q}_s/\dot{Q}_t$ is constant, P_{aO_2}/P_{AO_2} must vary as F_{IO_2} changes. If P_{aO_2}/P_{AO_2} is constant $\dot{Q}_s/\dot{Q}_t$ must vary with changes in F_{IO_2}. In hemodynamically stable patients, EVA is a good estimate of true $\dot{Q}_s/\dot{Q}_t$. CONCLUSIONS: The O_2-tension-based indices are poor indicators of true $\dot{Q}_s/\dot{Q}_t$. Using bias and limits of agreement, the computer model accurately predicted the observed $\dot{Q}_s/\dot{Q}_t$ in critically ill patients. [Respir Care 1996;41(7):586-594.]

Background

Because the measurement and calculation of the physiologic shunt fraction, ($\dot{Q}_s/\dot{Q}_t$, or venous admixture), involve an invasive procedure, clinicians have attempted to develop an accurate bedside method of estimating $\dot{Q}_s/\dot{Q}_t$. The primary gas-exchange indices used to estimate $\dot{Q}_s/\dot{Q}_t$ (ratio of the cardiac output that does not participate in gas exchange to the total cardiac output) are the alveolar-arterial O_2-tension difference $[P_{(A-a)O_2}]$, the ratio of arterial-to-alveolar O_2 tensions (P_{aO_2}/P_{AO_2}), the ratio of arterial O_2 tension to the fraction of inspired O_2 (P_{aO_2}/F_{IO_2}), estimated venous admixture (EVA), and respiratory index (RI). (See the Glossary for terms used in this paper.)

Covelli et al[1] studied all of the O_2-tension-derived indices as estimates of $\dot{Q}_s/\dot{Q}_t$ in 33 patients with ARDS and calculated the correlation coefficients for each index against $\dot{Q}_s/\dot{Q}_t$. The correlations in order of best to worst were: P_{aO_2}/F_{IO_2} ($r = 0.90$), P_{aO_2}/P_{AO_2} ($r = 0.88$), RI ($r = 0.87$), and $P_{(A-a)O_2}$ ($r = 0.68$). Therefore, they reported that P_{aO_2}/F_{IO_2} was the best estimate for $\dot{Q}_s/\dot{Q}_t$. They also reported that if the difference between arterial and mixed-venous O_2 content $[C_{(a-\bar{v})O_2}]$ was between 3.3 and 6.0 mL/dL, r was 0.94 for P_{aO_2}/F_{IO_2}. Changes in $\dot{Q}_t$, O_2 consumption ($\dot{V}_{O_2}$), and $C_{(a-\bar{v})O_2}$ all affect $\dot{Q}_s/\dot{Q}_t$ and, therefore, the O_2-tension-derived indices of shunt. How-

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The author has no financial interest in any product involved in this study.

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Glossary of Terms Used in the Models

Abbreviation	Definition	Normal Value/Units
B	Solubility of O_2 in blood	0.0031 mL/dL/torr
C_{aCO_2}	Arterial CO_2 content	mL/dL; see equations
C_{aO_2}	Arterial O_2 content	mL/dL; see equations
$C_{(a-\bar{v})O_2}$	Arterial-mixed-venous O_2-content difference	mL/dL
$C_{\bar{v}CO_2}$	Mixed venous CO_2 content	mL/dL; see equations
$C_{\bar{v}O_2}$	Mixed venous O_2 content	mL/dL; see equations
EAV	Estimated arterial-venous O_2-content difference	5.0 mL/dL
EVA	Estimated venous admixture—an estimated or modified calculation of venous admixture (physiologic shunt)	See equations
f	Respiratory rate	12 breaths/min
Hb	Hemoglobin concentration	14.7 g/dL
Hct	Hematocrit	3(Hb); mL/dL
P_{ACO_2}	Alveolar CO_2 tension	torr; see equations
P_{aCO_2}	Arterial CO_2 tension	35-45 torr
P_{AO_2}	Alveolar O_2 tension	torr; see equations
P_{aO_2}	Arterial O_2 tension	85-95 torr
P_{aO_2}/P_{AO_2}	P_{aO_2}-P_{AO_2} ratio	See equations.
P_{aO_2}/F_{IO_2}	P_{aO_2}-F_{IO_2} ratio	See equations.
$P_{(A-a)O_2}$	Alveolar-arterial O_2-tension difference	torr; see equations
P_B	Barometric pressure	760 torr (at sea level)
$P_{\bar{v}CO_2}$	Mixed-venous carbon dioxide tension	torr; see equations
$P_{\bar{v}O_2}$	Mixed-venous oxygen tension	torr; see equations
P_{H_2O}	Water vapor pressure	47 torr
P_{IO_2}	Inspired oxygen tension	159 torr (F_{IO_2} 0.21)
$\dot{Q}_s/\dot{Q}_t$	Physiologic shunt fraction, or venous admixture	0.02
$\dot{Q}_t$	Cardiac output	5.0 L/min
R	Respiratory exchange ratio	0.8
RI	Respiratory index	$P_{(A-a)O_2}/P_{aO_2}$
T	Body temperature	37 °C
V_D	Dead space volume	0.150 L/breath
V_T	Tidal volume	0.510 L/breath
$\dot{V}_A$	Alveolar minute ventilation	L/min; see equations
$\dot{V}_{CO_2}$	CO_2 production	$\dot{V}_{O_2}(R)$ (mL/min)
$\dot{V}_{O_2}$	O_2 consumption	250 mL/min

ever, correlation is not the most appropriate statistical analysis for these data. Correlation shows only whether a relationship exists between two variables. But how well one method estimates the true value of a variable is better assessed using the mean difference between the two variables (bias) and the standard deviation of this difference (precision, or limits of agreement).[2]

The purpose of my study was to develop a mathematical model of steady-state gas exchange and to use the model to develop a computer simulation that would document the variability of the O_2-tension-based clinical indices of gas exchange as predictors of $\dot{Q}_s/\dot{Q}_t$. The computer model was designed to take into account various factors that had not been included in previous gas-exchange models. These included the inter-

action of the CO_2 and O_2 dissociation curves and the inclusion of acid-base and pH calculations.

Methods

Model Assumptions

The basic assumptions of this computer model simulation were (1) all causes of gas exchange impairment (ie, anatomic shunt, diffusion defect, and ventilation-perfusion mismatch) are included together as physiologic shunt, and (2) tissue gas exchange is lumped into a single compartment with a set constant $\dot{V}_{O_2}$. Distribution effects and possible tissue gas-exchange abnormalities are not included.

Two models were programmed for this study using the Simulation Control Program (SCoP) simulation language (v 3.4, 1991, Simulation Resources Inc, Barrien Springs MI). Model 1 included $\dot{Q}_s/\dot{Q}_t$ as a constant and F_{IO_2} as the independent variable. Model 2 included F_{IO_2} as a constant and $\dot{Q}_s/\dot{Q}_t$ as the independent variable.

SEE RELATED OVERVIEW ON PAGE 607.

Model Development—Basic Equations

The basic equations and program sequence are shown in Appendix 1. The respiratory exchange ratio (R) and $\dot{V}_{O_2}$ are used to calculate carbon dioxide production ($\dot{V}_{CO_2}$) (Equation 1). The partial pressure of water vapor (P_{H_2O}) is calculated using the equation developed by Ruiz et al[3] in 1975 (Equation 2). Alveolar minute ventilation ($\dot{V}_A$) is calculated from the respiratory rate (f), tidal volume (V_T), and the dead space volume (V_D) as shown in Equation 3. The alveolar CO_2 tension (P_{ACO_2}) is calculated from $\dot{V}_{CO_2}$ and alveolar minute ventilation ($\dot{V}_A$) (Equation 4). P_{IO_2} (inspired O_2 tension) is calculated from F_{IO_2}, barometric pressure (P_B), and P_{H_2O} (Equation 5). The alveolar-gas equation is used to calculate P_{AO_2} (Equation 6).

P_{ACO_2} and P_{AO_2} are used in a special routine to solve 15 simultaneous equations relating acid-base balance, alveolar gas tensions, end-capillary blood-oxygen ($C_{c'O_2}$) and carbon dioxide ($C_{c'CO_2}$) contents, and the interaction of the O_2 and CO_2 dissociation curves.

$C_{c'O_2}$ and $C_{c'CO_2}$ are the output variables from the 15 simultaneous equations and are then used to calculate C_{aO_2} and carbon dioxide content (C_{aCO_2}) (Equations 7 & 8). C_{aO_2} and C_{aCO_2} and a second set of the 15 simultaneous equations for arterial acid-base balance and dissociation curves are used to calculate P_{aO_2} and P_{aCO_2}.

C_{aO_2} and C_{aCO_2} are then used to calculate the mixed-venous oxygen ($C_{\bar{v}O_2}$) and carbon dioxide ($C_{\bar{v}CO_2}$) contents (Equations 9 & 10). $C_{\bar{v}O_2}$ and $C_{\bar{v}CO_2}$ and a third set of the 15 simultaneous equations for mixed-venous acid-base balance and dissociation curves are used to calculate the mixed-venous oxygen ($P_{\bar{v}O_2}$) and carbon dioxide ($P_{\bar{v}CO_2}$) tensions.

The P_{50} is defined as the oxygen tension at which the hemoglobin is 50% saturated with oxygen. The final basic calculation is to calculate the in-vivo P_{50} using the equations of Snyder and Pinsky.[4]

Model Development—Simultaneous Equations

Although many different mathematical descriptions[5-21] have been developed for the O_2 dissociation curve, this model uses the equations of Gomez[5] because of the excellent statistical fit to the patient data of Severinghaus.[21] Shifts in the oxyhemoglobin dissociation curve due to pH, temperature, and P_{aCO_2} are easy to include in the mathematical description (Equations 14-18).

Equations 21 and 22 for pH and base excess (BE) are from Siggaard-Andersen.[22] The remaining equations used to describe the CO_2 dissociation curve are from Kelman[23] (Equations 19-28).

The same 15 simultaneous equations are used 3 different times in the computer model—for pulmonary end-capillary blood, for arterial blood, and for mixed-venous blood.

Equations 29-33 in Appendix 1 were used to derive the 5 gas-exchange indices calculated as part of the model analysis—$P_{(A-a)O_2}$, P_{aO_2}/P_{AO_2}, P_{aO_2}/F_{IO_2}, EVA, and RI.

Clinical Validation

The computer model was validated by collecting hemodynamic and gas exchange data from 37 patients, with a total of 50 data points. The patient population consisted of all critically ill patients in two intensive care units in Louisiana. The patients selected had flow-directed pulmonary artery catheters already in place for hemodynamic and gas exchange monitoring. These patient data were collected after receiving institutional review board approval for data collection. Patient consent was not required by the institutional review board because the data were already being collected from these patients as part of their daily care. Each patient was studied at least once and some were studied twice. Data from a previously studied patient were collected 48 hours or later after the first data collection.

The data collected consisted of arterial and mixed-venous blood gas values and measured $\dot{Q}_t$. C_{aO_2} and C_{aCO_2} were calculated from the blood-gas values, and $C_{(a-\bar{v})O_2}$ was calculated. $\dot{V}_{O_2}$ was calculated from $\dot{Q}_t$ and $C_{(a-\bar{v})O_2}$. R was assumed to be 0.8, and $\dot{V}_{CO_2}$ was calculated using Equation 1. P_{ACO_2} was assumed equal to P_{aCO_2} from the arterial blood-gas results. $\dot{V}_A$ was calculated from a rearrangement of Equation 4. The patient's P_{AO_2} was calculated using Equations 5 and 6.

$$\dot{Q}_s/\dot{Q}_t = (C_{c'O_2} - C_{aO_2})/(C_{c'O_2} - C_{\bar{v}O_2}).$$

These values were then entered into the computer model to predict $\dot{Q}_s/\dot{Q}_t$ and P_{aO_2} for each patient. The $\dot{Q}_s/\dot{Q}_t$ for each patient was calculated from the classic shunt fraction equation and compared to that predicted by the computer model.

The observed $\dot{Q}_s/\dot{Q}_t$ and P_{aO_2} were compared to the predicted values from the computer model. The Bland and Altman[2] limits of agreement procedure was used to calculate the bias and precision between the observed values and those predicted by the computer model.

Results

Effects of F_{IO_2} on Indices of $\dot{Q}_s/\dot{Q}_t$

The first part of the study involved looking at the effects of changing F_{IO_2} on the different gas exchange indices (Model 1). The plot in Figure 1A shows $P_{(A-a)O_2}$ versus F_{IO_2} for 3 values of $\dot{Q}_s/\dot{Q}_t$. This graph shows that changing F_{IO_2} greatly affects the $P_{(A-a)O_2}$ at any $\dot{Q}_s/\dot{Q}_t$. The graph in Figure 1B shows the effect of changing F_{IO_2} on the P_{aO_2}/P_{AO_2} at 3 $\dot{Q}_s/\dot{Q}_t$ values. The magnitude of the change in P_{aO_2}/P_{AO_2} is less than that in the $P_{(A-a)O_2}$. However, for any given $\dot{Q}_s/\dot{Q}_t$ value, the P_{aO_2}/P_{AO_2} varies widely.

Figure 2A shows the relationship between P_{aO_2}/F_{IO_2} and F_{IO_2} for 3 $\dot{Q}_s/\dot{Q}_t$ values. Again, the change is smaller in magnitude than that seen with the comparison to $P_{(A-a)O_2}$, but the difference is important. For example, if $\dot{Q}_s/\dot{Q}_t$ is 0.15 and F_{IO_2} varies between 0.21 and 1.0, then P_{aO_2}/F_{IO_2} is seen to start at about 325, drop to 250 at an F_{IO_2} of 0.5, and then increase to a value of about 400 at an F_{IO_2} of 1.0. The plot in Figure 2B shows the relationship between the RI and F_{IO_2} and that this relationship is complex.

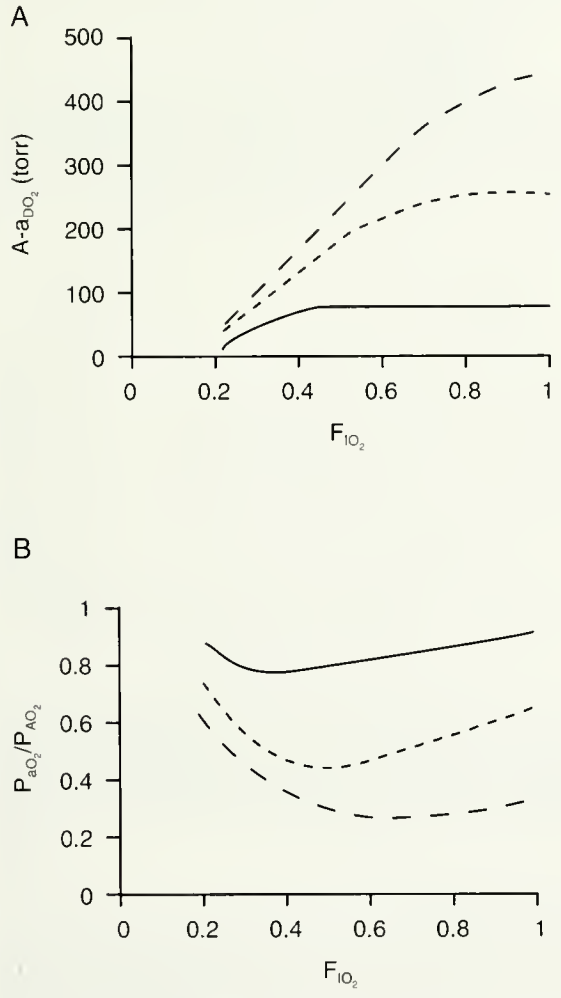

Fig. 1. Computer model of the effects of F_{IO_2} on (A) the alveolar-arterial P_{O_2} difference and (B) the P_{aO_2}-P_{AO_2} ratio at different levels of physiologic shunt—$\dot{Q}_s/\dot{Q}_t$—0.05 (—), 0.15 (- - -), 0.25 (– –).

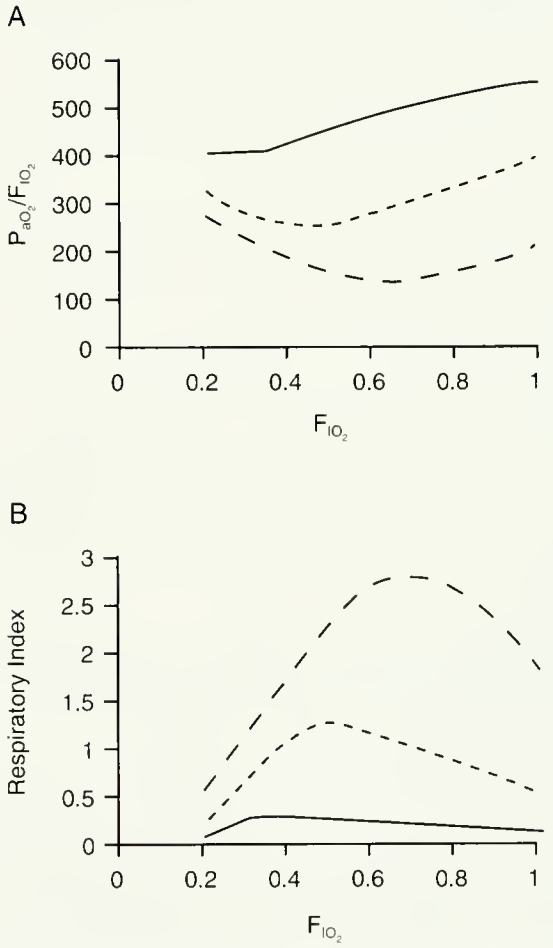

Fig. 2. Computer model of the effects of F_{IO_2} on (A) the P_{aO_2}-F_{IO_2} ratio and (B) the respiratory index ($[P_{aO_2}/P_{AO_2}]/F_{IO_2}$) at different levels of physiologic shunt—$\dot{Q}_s/\dot{Q}_t$—0.05 (—), 0.15 (- - -), 0.25 (– –).

Figure 3A shows the effect of changing F_{IO_2} on the P_{aO_2}/P_{AO_2} at 4 different levels of shunt. Notice that if shunt is constant then P_{aO_2}/P_{AO_2} changes as F_{IO_2} is varied. For exam-

ple, at a shunt of 0.12, P_{aO_2}/P_{AO_2} decreases as F_{IO_2} changes from 0.21 to 0.4. The P_{aO_2}/P_{AO_2} remains constant as F_{IO_2} changes from 0.4 to about 0.55, then increases as F_{IO_2} increases to 1.0.

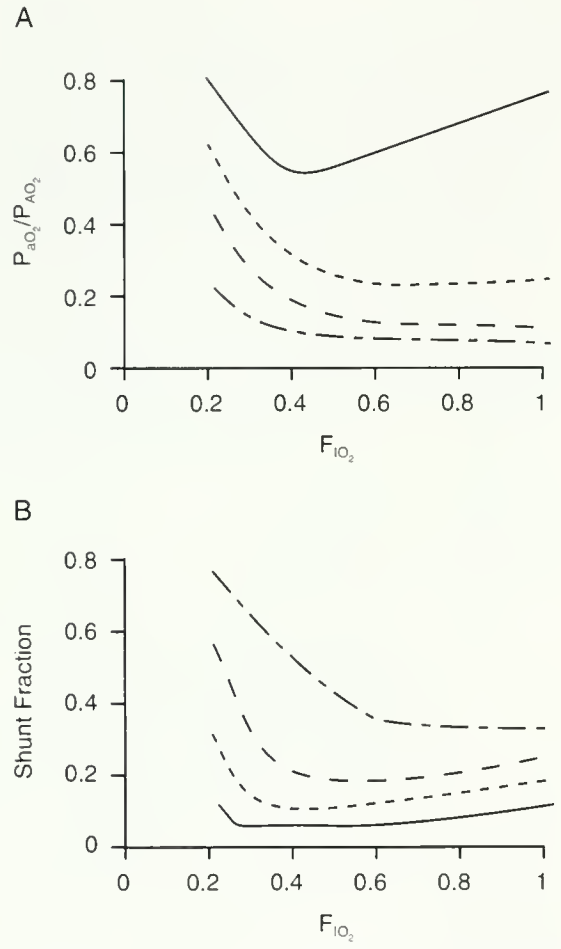

Fig. 3. Computer model of the effects of F_{IO_2} on (A) P_{aO_2}-P_{AO_2} ratio at different constant shunt levels [0.12 (—), 0.30 (- - -), 0.55 (– –), 0.76 (– - –)] and (B) physiologic shunt for different constant values of P_{aO_2}-P_{AO_2} ratio [0.8 (—), 0.6 (- - -), 0.4 (– –), 0.2 (– - –)].

One of the most interesting relationships found with this computer simulation is shown in Figure 3B. This graph shows the effect of changing F_{IO_2} on $\dot{Q}_s/\dot{Q}_t$ when P_{aO_2}/P_{AO_2} is held constant. If P_{aO_2}/P_{AO_2} is constant then $\dot{Q}_s/\dot{Q}_t$ is seen to decrease as F_{IO_2} increases from 0.21 to 0.4. The $\dot{Q}_s/\dot{Q}_t$ then remains constant as F_{IO_2} changes from 0.4 to 0.55, then increases slightly as F_{IO_2} increases to 1.0.

Indices To Predict Shunt

In Model 2, the F_{IO_2} was entered as a constant and $\dot{Q}_s/\dot{Q}_t$ was the independent variable. The model was programmed to look at the effects of changes in the different constants on the relationship between $\dot{Q}_s/\dot{Q}_t$ and the various gas-exchange indices. The constants studied were F_{IO_2}, $\dot{Q}_t$, hemoglobin concentration (Hb), $\dot{V}_{O_2}$, P_B, R, V_T, V_D, f, and body temperature (T).

Figure 4 shows the relationship between $\dot{Q}_s/\dot{Q}_t$ and EVA. The $C_{(a-\bar{v})O_2}$ was assumed to be 5.0 mL/dL in this simulation. Figure 4A shows that $\dot{Q}_t$ has a large effect on this relationship. Figure 4B shows that this relationship is not affected by changes in F_{IO_2}.

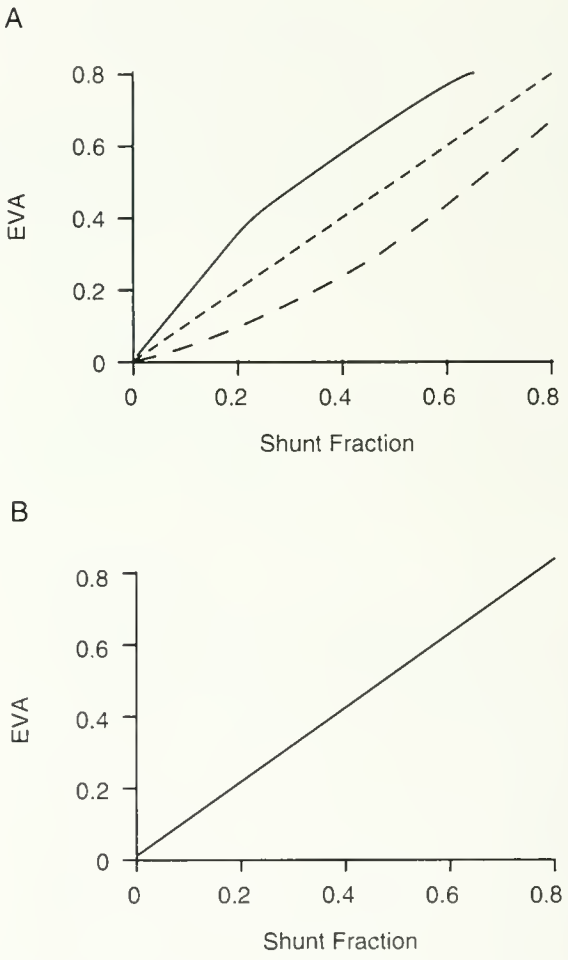

Fig. 4. Computer model showing the relationship between estimated venous admixture (EVA) and physiologic shunt fraction with (A) changes in cardiac output [2.5 (—), 5.0 (- - -), 10.0 (– –)] and (B) changes in F_{IO_2} [0.21 (—), 0.5 (- - -), 1.0 (– –)].

Clinical Validation of the Model

Figure 5 shows the bias-and-precision plot for the observed $\dot{Q}_s/\dot{Q}_t$, measured in 37 patients (50 measurements) against the predicted shunt based on the computer model. Bias (precision) is 0.0012 (0.0244)—not significantly different from zero. The plot in Figure 6 shows the bias-and-precision plot for the

observed P_{aO_2} against the P_{aO_2} predicted from this computer model, from the same patients. Bias (precision) is 3.7 (3.99) and, although significantly different from zero, is probably not clinically important. The plot also shows that the higher the P_{aO_2}, the more the computer model underestimates the true P_{aO_2}, particularly when true P_{aO_2} is > 100 torr.

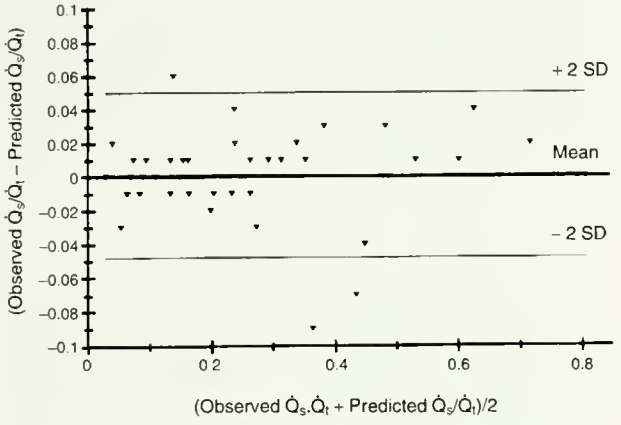

Fig. 5. Plot of the bias and limits of agreement between the observed physiologic $\dot{Q}_s/\dot{Q}_t$ from patient data and $\dot{Q}_s/\dot{Q}_t$ predicted by the computer model, where bias = 0.0012, SD = 0.0244, t = 0.3480, and p = 0.729.

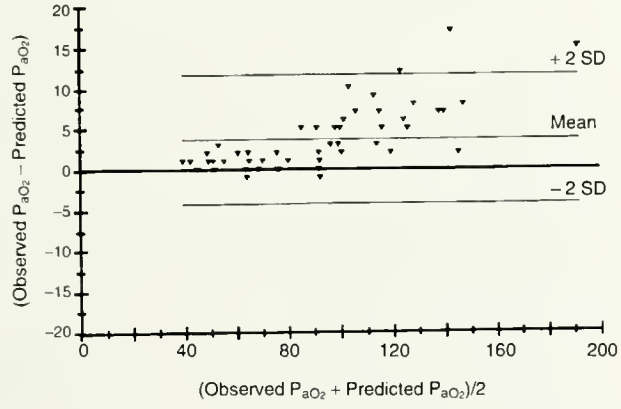

Fig. 6. Plot of the bias and limits of agreement between the observed P_{aO_2} from patient data and the P_{aO_2} predicted by the computer model, where bias = 3.70, SD = 3.9911, t = 6.56, and p < 0.001.

Figure 7 shows the polynomial regression for the P_{aO_2}/P_{AO_2} against the observed $\dot{Q}_s/\dot{Q}_t$ from the patient data.

$$\dot{Q}_s/\dot{Q}_t = e^{[a + b(P_{aO_2}/P_{AO_2})]},$$

where, a = –0.289211 and b = –3.5477.

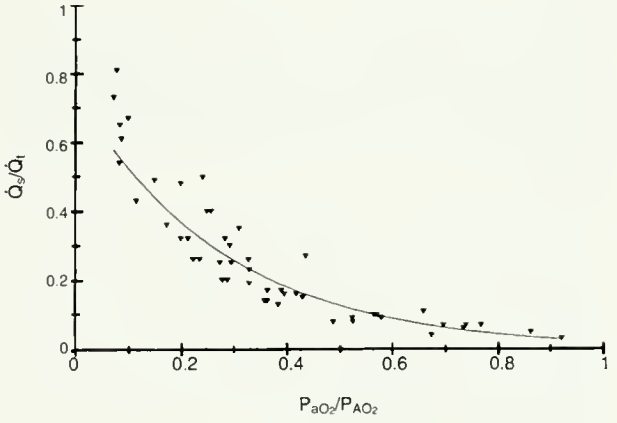

Fig. 7. Polynomial regression of $\dot{Q}_s/\dot{Q}_t$ as a function of P_{aO_2}/P_{AO_2} from patient data, where $\dot{Q}_s/\dot{Q}_t = e^{[a+b(P_{aO_2}/P_{AO_2})]}$, a = –0.289211, b = –3.5477, and r = –0.953.

The plot in Figure 8 shows the polynomial regression for the P_{aO_2}/F_{IO_2} plotted against the observed $\dot{Q}_s/\dot{Q}_t$.

$$\dot{Q}_s/\dot{Q}_t = e^{[a + b(P_{aO_2}/F_{IO_2})]},$$

where a = –0.169036 and b = –0.006591.

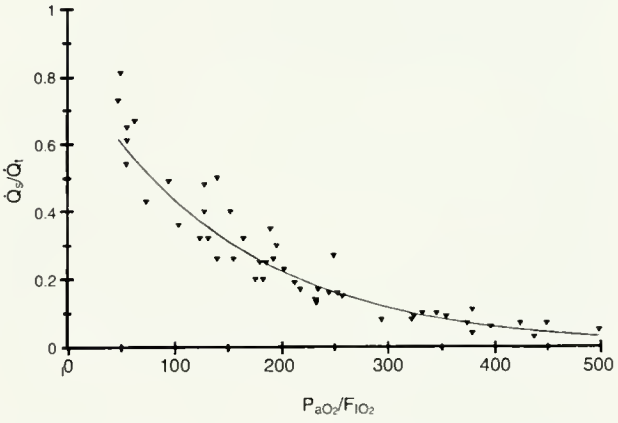

Fig. 8. Polynomial regression of $\dot{Q}_s/\dot{Q}_t$ as a function of P_{aO_2}/F_{IO_2} from patient data, where $\dot{Q}_s/\dot{Q}_t = e^{[a+b(P_{aO_2}/F_{IO_2})]}$, a = –0.169036, b = -0.006591, and r = –0.949.

The data in these two plots are from the patient data and not from the computer model.

Discussion

The purpose of this paper was to develop a mathematical model of steady-state pulmonary gas exchange and to use this model to investigate the variability of the various O_2-tension-based clinical indices of gas exchange commonly used. Many of the graphs from this model are very similar to others pub-

lished previously.[24-40] However, in these previously published models the equations and interactions were not delineated.

In this model, the effect of the interaction between the O_2 and CO_2 dissociation curves and the acid-base relationships for end-capillary, arterial, and mixed-venous blood are included in the model. To my knowledge this is the first model to include these relationships.

The unexpected results generated by this model are shown in Figure 3. The plot in Panel A shows that P_{aO_2}/P_{AO_2} varies directly with changes in F_{IO_2} when $\dot{Q}_s/\dot{Q}_t$ is held constant. Panel B shows that $\dot{Q}_s/\dot{Q}_t$ varies with changes in F_{IO_2} when P_{aO_2}/P_{AO_2} remains constant. This means that, in those patients whose P_{aO_2}/P_{AO_2} remains constant as F_{IO_2} is changed, $\dot{Q}_s/\dot{Q}_t$ must be changing and P_{aO_2}/P_{AO_2} is not a good indicator of the magnitude of the gas-exchange abnormality. Also, in the clinical population in whom the P_{aO_2}/P_{AO_2} is seen to change with changes in F_{IO_2}, the $\dot{Q}_s/\dot{Q}_t$ must be remaining constant as seen in Fig. 3A. Viale et al[41] and Gilbert[42] both reported this finding in their studies and concluded that when P_{aO_2}/P_{AO_2} remains constant and F_{IO_2} is varied, $\dot{Q}_s/\dot{Q}_t$ is actually changing substantially.

The only index studied here that was not affected by F_{IO_2} was EVA, assuming a 5 mL/dL $C_{(a-\bar{v})O_2}$ (Fig. 4B). However, this index is significantly affected by changes in $\dot{Q}_t$ (Fig. 4A). This is in agreement with the results of Hess et al[43] who found that the EVA is a good estimate of the true shunt in patients who are hemodynamically stable.

The limits of agreement between the observed and model-predicted $\dot{Q}_s/\dot{Q}_t$ and patient-derived P_{aO_2} are acceptable (Figs. 5 & 6). However, the model tended to underestimate the observed P_{aO_2} at greater than 100 torr (Fig. 6). This underestimation is probably because the computer model only dealt with a fixed $\dot{Q}_s/\dot{Q}_t$ and not with ventilation-perfusion mismatching. In the patients studied, ventilation-perfusion mismatching is more responsive to increasing F_{IO_2} than is a fixed $\dot{Q}_s/\dot{Q}_t$. This analysis suggests that $\dot{Q}_s/\dot{Q}_t$ predicted by this computer model was not substantially different from the observed values measured in the patients.

The polynomial regressions shown in Figures 7 and 8 give prediction equations that may be useful at the bedside to more accurately predict the true $\dot{Q}_s/\dot{Q}_t$ from the P_{aO_2}/P_{AO_2} or the P_{aO_2}/F_{IO_2} values. Further study with a larger number of patients is needed to determine whether these prediction equations are clinically useful.

Conclusions

The computer model simulation of pulmonary gas exchange resulted in the following major findings:

- In the clinical observations of a constant P_{aO_2}/P_{AO_2}, $\dot{Q}_s/\dot{Q}_t$ must actually be changing as F_{IO_2} is varied.
- Oxygen-tension-based indices of gas exchange are poor indicators of the true $\dot{Q}_s/\dot{Q}_t$.
- In patients who are hemodynamically stable, EVA is a good estimate of the true $\dot{Q}_s/\dot{Q}_t$ and is not affected by changes in F_{IO_2}.
- The computer model simulation accurately predicts the observed $\dot{Q}_s/\dot{Q}_t$ and P_{aO_2} in a group of critically ill patients.

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APPENDIX 1. Model Sequence and Equations

R and $\dot{V}_{O_2}$ are used to calculate carbon dioxide production:

$$\dot{V}_{CO_2} = R(\dot{V}_{O_2}). \quad [1]$$

P_{H_2O} is calculated using the equation developed by Ruiz et al[2] in:

$$P_{H_2O} = 0.4 + 10^{(T + 0.7659[0.0244])}. \quad [2]$$

$\dot{V}_A$ is calculated from f, V_T, and V_D:

$$\dot{V}_A = f(V_T - V_D). \quad [3]$$

P_{ACO_2} is calculated from the $\dot{V}_{CO_2}$ and $\dot{V}_A$:

$$P_{ACO_2} = (0.863[\dot{V}_{CO_2}])/\dot{V}_A. \quad [4]$$

P_{IO_2} is calculated from the F_{IO_2}, P_B, and P_{H_2O}:

$$P_{IO_2} = F_{IO_2}(P_B - P_{H_2O}). \quad [5]$$

The alveolar gas equation is used to calculate P_{AO_2}:

$$P_{AO_2} = (P_{IO_2} - [P_{ACO_2}/R]) + P_{ACO_2} \cdot F_{IO_2}([1 - R]/R). \quad [6]$$

$C_{c'O_2}$ and $C_{c'CO_2}$ are the output variables from the 15 simultaneous equations and are used to calculate C_{aO_2} and C_{aCO_2}:

$$C_{aO_2} = C_{c'O_2} + (\dot{Q}_s/\dot{Q}_t/[1 - \dot{Q}_s/\dot{Q}_t])(\dot{V}_{O_2}/[10]\dot{Q}_t). \quad [7]$$

$$C_{aCO_2} = C_{c'CO_2} + (\dot{Q}_s/\dot{Q}_t/[1 - \dot{Q}_s/\dot{Q}_t])(\dot{V}_{CO_2}/[10]\dot{Q}_t). \quad [8]$$

C_{aO_2} and C_{aCO_2} plus another 15 simultaneous equations for arterial acid-base balance and dissociation curves are used to calculate P_{aO_2} and P_{aCO_2}. C_{aO_2} and C_{aCO_2} are then used to calculate $C_{\bar{v}O_2}$ and $C_{\bar{v}CO_2}$:

$$C_{\bar{v}O_2} = C_{aO_2} - (\dot{V}_{O_2}/[10]\dot{Q}_t). \quad [9]$$

$$C_{\bar{v}CO_2} = C_{aCO_2} - (\dot{V}_{CO_2}/[10]\dot{Q}_t). \quad [10]$$

$C_{\bar{v}O_2}$ and $C_{\bar{v}CO_2}$ are combined with 15 simultaneous equations for mixed-venous acid-base balance and dissociation curves $P_{\bar{v}O_2}$ and $P_{\bar{v}CO_2}$. The following 3 equations are calculated in the initial section of the computer model:

$$\alpha = 0.037 + 0.00057\ (37 - T) + 0.00002\ (37 - T)^2, \quad [11]$$

where T is the temperature in degrees Celsius.

$$Hct = (3)\ Hb, \quad [12]$$

where Hct is the percentage of the volume of blood that is cells, and Hb is the blood hemoglobin concentration in g/dL.

$$h = Hct\ (0.01), \quad [13]$$

where, h is the hematocrit expressed as a fraction.

The following 5 equations describe the O_2 dissociation curve and are the first 5 of the 15 simultaneous equations:

$$P_{Corr} = P_{aO_2}\ (10^Z), \quad [14]$$

$$Z = 0.024(37-T) + 0.4(pH-7.4) + 0.06(\log\ [40]-\log\ [P_{aCO_2}]), \quad [15]$$

$$U = A1(P_{Corr}) + A2(P_{Corr})^2 + A3(P_{Corr})^3, \quad [16]$$

$$= U/1 + U, \text{ and} \quad [17]$$

$$C_{aO_2} = (1.39\ [Hb])\ (S_{aO_2} + (B\ P_{aO_2}), \quad [18]$$

where P_{Corr} = the corrected P_{aO_2} based on pH, T, and P_{aCO_2}; Z = correction factor for causes of shifts in the oxygen dissociation curve, S_{aO_2} = oxyhemoglobin saturation; and B = 0.0031, a constant for dissolved O_2.

The following 10 equations relate the CO_2 dissociation curve and acid-base equations. These equations are the last 10 of the 15 simultaneous equations:

$$pK = 6.086 + 0.042\ (7.4 - pH) + (38 - T)\ (0.0047 + [0.0014\ (7.4 - pH)]) \quad [19]$$

$$HCO_3^- = \alpha - P_{aCO_2}(10)^{[pH - pK]}. \quad [20]$$

$$pH = 7.4 - [(HCO_3^- - 24) - (BE/BX)]/BZ, \quad [21]$$

where BE = base excess; BX = 1 − (0.0143 Hb); and BZ = 9.5 + (1.63 Hb).

$$BE = [(1 - ctHb)\ (0.023)\ (HCO_3^- - 24)] + [(2.30[ctHb + 7.7])\ (pH - 7.40)], \quad [22]$$

where ctHb = Hb(0.6205783) to change units from g/dL to mmol/L).

$$C_{aCO_2}\ (Plasma) = \alpha P_{aCO_2}\ (1 + 10^{[pH - pK]}). \quad [23]$$

$$DOX^* = 0.590 + [(0.2913)(7.4 - pH)] - [(0.0844)(7.4 - pH)^2]. \quad [24]$$

$$DR = 0.664 + [(0.2275)\ (7.4 - pH)] - [(0.0938)(7.4 - pH)^2]. \quad [25]$$

$$D = DOX + [(DR - DOX)(1 - S_{aO_2})]. \quad [26]$$

$$CC = DC_{aCO_2}\ (plasma). \quad [27]$$

$$C_{aCO_2}\ (Total) = (2.226)([(h)(CC)]) + [(1 - h)(C_{aCO_2})](plasma). \quad [28]$$

*DOX, DR, and D are constants that are used to account for the effects of S_{aO_2} and pH on C_{aCO_2}.

The following are the gas-exchange indices calculated as part of the model validation:

$$a\text{-}A\ ratio = P_{aO_2}/P_{AO_2}. \quad [29]$$

$$P_{(A-a)O_2} = P_{AO_2} - P_{aO_2}. \quad [30]$$

$$P_{aO_2}\text{-}F_{IO_2}\ ratio = P_{aO_2}/F_{IO_2}. \quad [31]$$

$$RI = P_{(A-a)O_2}/P_{aO_2}. \quad [32]$$

$$EVA = (C_{c'O_2} - C_{aO_2})/[(C_{c'O_2} - C_{aO_2}) + (C_{aO_2} - C_{\bar{v}O_2})], \quad [33]$$

where $C_{c'O_2} - C_{aO_2} = 5.0$ mL/dL.

Proficiency among Respiratory Therapy Educators, Staff, and Students in Using Algorithms for Therapist-Driven Protocols

Rebecca L Meredith RRT, Susan P Pilbeam MS RRT,
Kevin McCarthy RCPT, and James K Stoller MD

BACKGROUND: Although therapist-driven protocols (TDPs) are becoming increasingly popular, successful implementation requires confidence in assessment skills and in implementing treatment algorithms. METHODS & MATERIALS: To assess the level of respiratory care practitioners' (RCP) preparedness for evaluate-and-treat programs, we undertook a survey in which 6 case studies of non-ICU, non-mechanically ventilated patients were mailed to 1,237 RCPs (960 students, 137 staff RCPs, 140 RCP instructors) in 62 schools and 10 hospitals. Using treatment algorithms from the Cleveland Clinic Foundation Respiratory Therapy Consult Service, respondents were asked to generate a care plan for each case study. 'Correct' answers were identified beforehand by expert consensus. The answers of individual respondents were graded against correct answers. RESULTS: Mean response rate was 47.8% (students 50.8%, staff 50.0%, instructors 24.2%). Mean (SD) rate of correct responses overall was 49.0 (18.6)%. Comparison of response rates by training level showed that instructors performed better than staff RCPs who performed better than students [64.1 (15.5)% vs 54.8 (18.2)% vs 47.5 (18.3)%, $p < 0.05$]. In contrast, rates of correct responses were no higher among students completing 4-year vs 2-year vs 1-year (technician) programs [44.9 (20.2)% vs 47.8 (18.4)% vs 47.8% (16.4)%, $p > 0.05$]. However, students training at clinical sites using TDPs scored better than their counterparts at hospitals not using TDPs. Although 73% of 434 responding students reported being comfortable with protocols, the correct response rate was also low, 47.0 (18.4)%. CONCLUSIONS: (1) Overall, the rate of correct responses was low in this study. (2) Among respondents, instructors demonstrated the highest frequency of correct responses, exceeding staff therapists and students. (3) No effect of length of training program was apparent, although overall student performance was low. (4) Among students, training at clinical sites using TDPs was associated with better performance in the case-study exercises. [Respir Care 1996;41(7):595-600]

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An abstract based on this study was presented at the RESPIRATORY CARE OPEN FORUM during the Annual Meeting of the American Association for Respiratory Care, in Nashville, Tennessee, December 11-14, 1993.

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Introduction

Therapist-driven protocols (TDPs),[1-4] also called patient-driven protocols, are becoming increasingly popular in respiratory care. For example, a recent poll of attendees at an American Association for Respiratory Care (AARC) House of Delegates meeting[5] showed that 40% of the hospitals represented were currently using protocols and an additional 15% were preparing to implement protocols. However, successful implementation of TDPs requires overcoming several obstacles[6] that may include resistance from hospital administration related to uncertainty about the efficacy and/or cost-effectiveness of TDPs; resistance from hospital nursing

service related to overlap of patient monitoring and care activities and to promoting other new models of care delivery (eg, patient-focused care); resistance from physicians; and resistance from respiratory therapists themselves related to complacency and/or lack of confidence in their own assessment skills. Therapists' uncertainty about assessment skills may derive from their preferring a traditional task-oriented identity or from their experience in curricula that underemphasize training and assessment skills in respiratory therapy.

Using proficiency in applying TDP algorithms as a measure of ability to develop appropriate care plans, we undertook to assess performance when respiratory care students, educators, and practitioners apply algorithms to case studies. We specifically examined whether students' performance using algorithms relates to the length and type of respiratory care curricula or to affiliation of the respiratory care program with clinical sites using TDPs. Furthermore, we examined the relationship between staff practitioners' performance and use of TDPs in their hospitals.

Methods & Materials

Invitations to participate in the current study were initially extended by telephone to 62 respiratory care schools selected at random from the 1991 edition of the list of schools in the Joint Review Committee for Respiratory Therapy Education Programs booklet.[7] The 10 participating hospitals were selected based on their willingness to participate and, in 3 instances (ie, Cleveland Clinic Foundation, Cleveland, Ohio; Robert Packer Hospital, Sayre, Pennsylvania; and Lutheran Medical Center, Wheat Ridge, Colorado) specifically because they were known to have used TDPs for at least 1 year before the study began. Of the 10 hospitals sampled, 5 were using TDPs (TDP hospitals) and 3 were not; 2 hospitals did not respond. Responses were received from 80% of the TDP hospitals and 100% of the non-TDP hospitals.

Mailings to participating institutions included packets containing a letter of introduction, a statement of the study purpose, directions for completion of the materials, and a packet for each participant. A total of 1,237 individual-participant packets were distributed. Each packet included a questionnaire regarding descriptive data (program type and length, location, employment status, and degree of comfort with one's own assessment skills), 6 case studies of non-ICU (intensive care unit) patients (3 from the Cleveland Clinic Foundation and 3 from Lutheran Medical Center), and algorithms to help guide the care plans. The cases that were provided were in current use for staff education at the contributing institutions (Appendix 1). The respiratory care algorithms that were distributed were based on the Clinical Practice Guidelines from the American Association for Respiratory Care[8-12] and were developed by the Section of Respiratory Therapy at the Cleveland Clinic Foundation.[3]

'Correct' answers for each case study were determined in advance by expert consensus. The group of experts included 5 program instructors, 9 experienced respiratory therapists, and 2 pulmonary physicians. The answers of individual respondents were graded against the correct answers, and results were reported as percentage of total correct responses. Appendix 1 presents an example of a case study, the relevant algorithms, and the correct answers.

Statistical analysis was performed using SIGMASTAT, Version 1.02 (Jandel Scientific, San Rafael CA) with the Mann-Whitney Rank Sum test for nonparametric distributions and the unpaired *t* test for normally distributed data. Comparisons with $p < 0.05$ were considered statistically significant. Continuous variables are summarized as mean ± standard deviation, or mean (SD).

Results

Rates of response to the mailings are shown in Figure 1. Of the 488 students responding (all of whom were within 2 months of graduating from an American Medical Association-approved respiratory care program), 75% (n = 368) were associate-degree candidates, 16% (n = 79) were technician-certificate candidates, and 8% (n = 41) were baccalaureate-degree candidates. Figure 2 shows the pattern of mailings and responses. Figure 3 presents the rate of correct responses among students, instructors, and staff therapists. The overall rate of correct responses was 49.0 (18.6)%. Among 315 student participants who reported being comfortable making independent decisions for respiratory care plans (64% of all student participants), the correct-response rate was 47.0 (18.4)%, which showed no statistically significant difference from the rate of correct responses from among less confident students, 48.3 (18.8)%, p = 0.424.

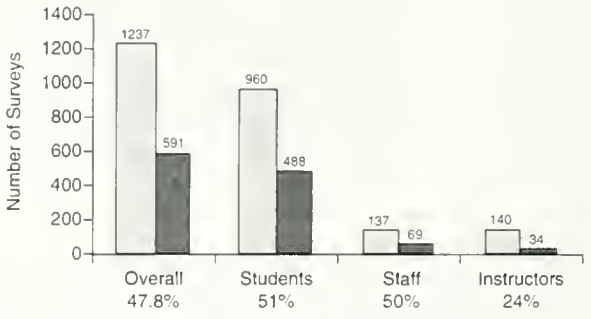

Fig 1. Light bars indicate the number of surveys sent and dark bars indicate the number returned. The percent response by respondent type is indicated on the abscissa.

Stratifying the rates of correct responses by the type of respiratory care program showed no differences among graduates of 4-year, 2-year, or 1-year (technician) programs (Fig. 4). Among the 69 staff respondents (37 of whom were staff therapists at the Cleveland Clinic Foundation), 50 worked in

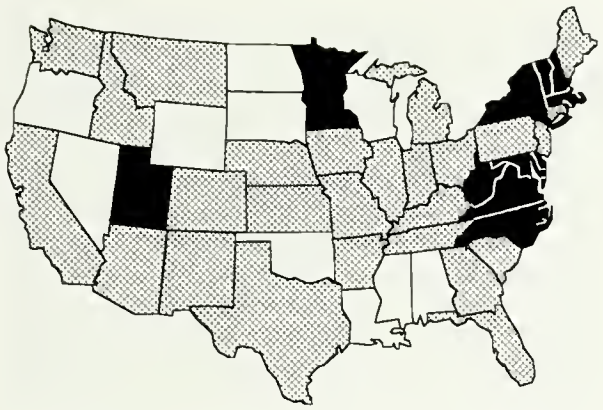

Fig. 2. States from which responses were received are shown with cross-hatched pattern, states from which responses were invited but not received are shown in black, and states that did not participate are shown in white. Questionnaires were sent to programs in 38 states and received from 26 states.

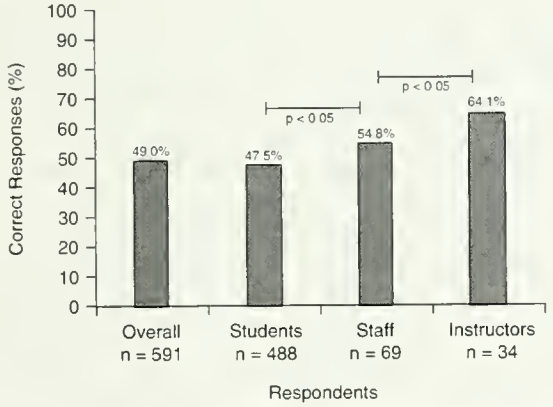

Fig. 3 Among the three groups sampled, instructors submitted the highest percent of correct responses, followed by staff, then by students. Number of individuals (n) on the abscissa represent the total number of members in each group. Differences between students and staff and between staff and instructors were significant at $p < 0.05$.

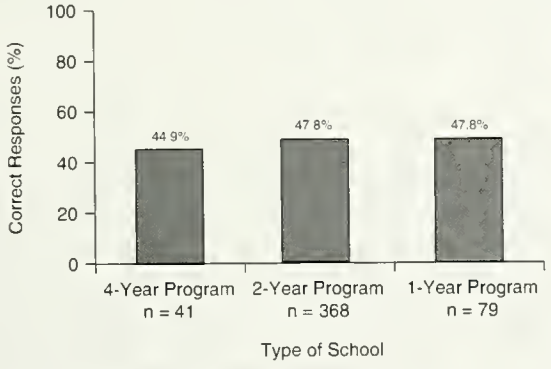

Fig. 4. No significant difference was observed between respondents trained in 4-year, 2-year, or 1-year respiratory care programs. The numbers of individuals (n) on the abscissa represent the total number of members of each group.

3 hospitals that had used TDPs for at least 1 year (so-called TDP hospitals), 13 worked in the 3 hospitals not using TDPs, and 6 worked in the 2 hospitals that had used TDPs for less than 1 year before the study began. As shown in Figure 5, the rate of correct responses was significantly higher among staff at TDP hospitals than among staff at non-TDP hospitals ($p < 0.001$). In a similar pattern, students at programs affiliated with TDP hospitals performed better than their counterparts (Fig. 6). Comparison of correct response rates among working versus nonworking students showed no difference (median response rate for working students = 64%; for nonworking students = 52%, $p = 0.272$).

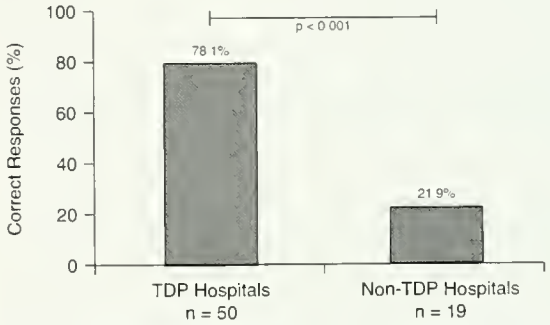

Fig. 5. The difference between rates of correct responses by staff therapists in hospitals using therapist-driven protocols (TDP hospitals) and those not using TDPs was significant at $p < 0.01$. The numbers of individuals (n) on the abscissa represent the total number of members of each group.

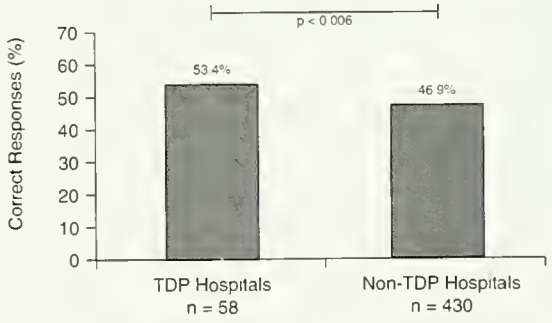

Fig. 6. The difference between rates of correct responses by students at therapist-driven protocols (TDP) hospitals and those at non-TDP hospitals is significant at $p < 0.0069$. The numbers of individuals (n) on the abscissa represent the total number of members of each group.

Discussion

The main findings of the current study are: (1) Study respondents achieved a low overall rate of correct responses [49.0 (18.6)%] when applying treatment algorithms to case studies. (2) Instructors had the highest rate of correct responses, exceeding staff therapists and students. (3) In univariate analyses, no

effect of length of students' training program or working status was apparent, but students in programs affiliated with TDP hospitals scored higher than their counterparts in non-TDP hospitals. In a similar pattern, staff working in TDP hospitals performed better than their counterparts in non-TDP hospitals. Taken together, these latter two findings suggest that exposure to TDPs, either during training or employment, enhances performance in applying algorithms to case studies.

Several explanations can be offered for the low rate of correct responses observed. First, if respondents failed to understand the written study instructions, imperfect performance with the case studies would result. Of potentially greater concern, if the case studies were incomplete and/or failed to provide sufficient information to allow care plans to be generated with the algorithms, the rate of correct responses would decrease. Although neither possibility can be discounted, we believe that the written instructions sent to each respondent were succinct and straightforward. Also, the case studies used in the study were specifically prepared to be inclusive and to allow a care plan to be deduced by applying the available algorithms. Case studies available from the National Board for Respiratory Care (NBRC) were not chosen for use in this study because they are available to educators and to students, thereby increasing the possibility that participants could be familiar with these cases. Also, because the NBRC clinical simulation problems were written for a different examination and without attention to these algorithms, use of NBRC cases would not assure providing enough information to allow the algorithms to be applied.

Another possible explanation for the low rate of correct responses is that respondents failed to understand the algorithms. Although this possibility also cannot be discounted, the branched logic format of the algorithms readily communicates both their purpose and their method of use. Because the study instructions were simple, the case study was inclusive, and the algorithms seemed straightforward and self-explanatory, we believe that the high rate of incorrect responses in this study at least partially reflects the inability of respondents to apply algorithms to clinical vignettes.

Our use of clinical information from case studies rather than from actual patient encounters was required by practical needs of this study (ie, to compare many respondents in a standard manner); but using case studies clearly limits the applicability of our findings to actual clinical assessment by therapists. Indeed, a reliable study of therapists' performance as assessors would include many methodologic features absent from our study, including direct observation of therapists conducting clinical assessments of actual patients whose respiratory care needs were recently assessed and were known to the examiners. Even when we concede the potential shortcomings of our data, the observation that students and staff in non-TDP hospitals scored lowest certainly invites concern that current training in assessment skills is inadequate and that greater emphasis in future curricula is needed.

It follows that educational institutions may benefit from incorporating the AARC Clinical Practice Guidelines[8-12] and algorithm-based study formats into their curricula, thereby providing more opportunities for students to develop patient care plans. Also, we believe that students should practice developing and defending respiratory care plans during rotations to clinical sites. For example, daily consult rounds at some institutions consist of supervisors' reviewing care plans with therapists, a format that we have found helpful in our institution to encourage discussion of respiratory care plans that have already been implemented. During this exercise, therapists must defend their care plans and are thereby encouraged to develop analytical skills.[13] Another strategy to help foster expertise in applying algorithms is to distribute actual patient case studies to staff members and to require respondents to submit algorithm-based respiratory care plans. The care plans are then graded against correct answers that are developed beforehand by expert consensus. In our experience, this exercise has proven useful to determine staff educational needs, to assess the ability of employees to develop appropriate care plans, and to assess individuals' performance serially over time. Similar training methods incorporating patient cases and use of algorithms may help students to improve their analytical thinking and problem-solving skills.[13,14]

Several other limitations of the current study must be considered when interpreting our results. First, the overall response rate to the questionnaires was only 47.8%, raising the possibility of participation bias. To the extent that participants were more likely to represent TDP hospitals, the rates of correct responses in the current study are likely to be inflated. This interpretation underscores our finding that proficiency in performing assessments was disappointing in this study.

Another shortcoming of the study is that proficiency in assessment skills was measured using case studies, which were used as surrogates for actual clinical encounters. As has been pointed out previously,[15] case studies may fail to simulate clinical reality in several important ways, including the unavailability of visual or auditory data in case studies, possible oversimplification, and the failure of case studies to include information about changes in clinical circumstances over time.

As a possible offset to factors that cause the proficiency ratings to be overestimated in this study, it should be emphasized that the algorithms used to generate respiratory care plans were previously unfamiliar to respondents. On this basis, we believe that respondents' performance would have been better if more extensive training in using the algorithms had been possible. Quality control studies at the Cleveland Clinic Foundation (in which we have serially assessed therapists' ability to develop an appropriate care plan using algorithms) suggest that proficiency in applying the algorithms improves over time and that the initial rates of successful use can be modest (unpublished data).

Conclusions

These data suggest that performance in generating respiratory care plans was poor overall when students, practicing therapists, and instructors were asked to apply algorithms to case studies. Instructors demonstrated the highest rate of correct responses, a predictable finding, but no effect of length of the training program was apparent. For both students and staff therapists, work experience with therapist-driven protocols was most strongly associated with better performance in generating correct care plans. Our findings have important implications for educators planning curricula and training-site affiliations for future respiratory care practitioners.

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Appendix 1:
Sample Case Study, 'Correct' Respiratory Care Plan, and Related Algorithms

A. Case Study:

A 66-year-old woman is admitted with decreasing mental status, shortness of breath and a 'moist' cough. She has a history of alcohol abuse, COPD, macrocytic anemia, cor pulmonale, an 80-pack-year history of smoking, and a nodule on the right lung. She is on oxygen at home (2 L/min by nasal cannula) receives ipratropium bromide (Atrovent) and albuterol (Proventil) via metered-dose inhaler (MDI).

Breath sounds: decreased bilaterally, rhonchi bilaterally, dyspnea on exertion

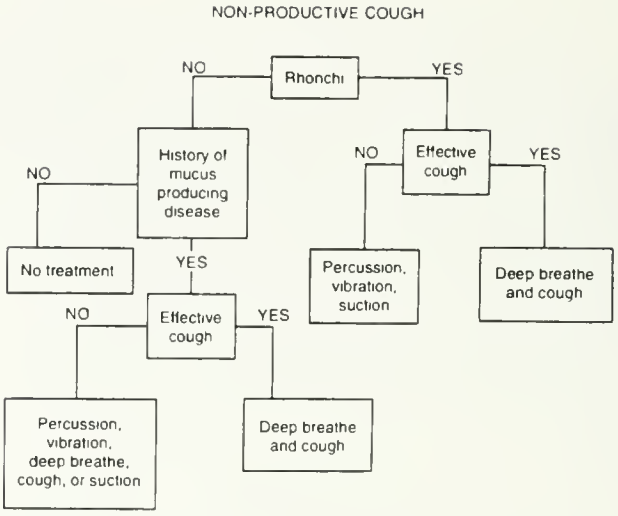
Cough: nonproductive and weak
Deep breath: unable to perform
Pulmonary function tests: unable to perform
Oxygenation: She is currently on 3 L/min of oxygen by nasal cannula with an S_{pO_2} of 90%. Her S_{pO_2} is 96% on 6 L/min nasal cannula
Arterial blood gas values: pH = 7.46, P_{aCO_2} = 32, P_{aO_2} = 54, S_{aO_2} = 90, bicarbonate = 22, Hb = 10.5 (on 3 L/min by cannula)
Vital signs: respiratory rate = 28, blood pressure = 126/70. heart rate = 104, temperature = 37° C
White blood cell count: 23,000

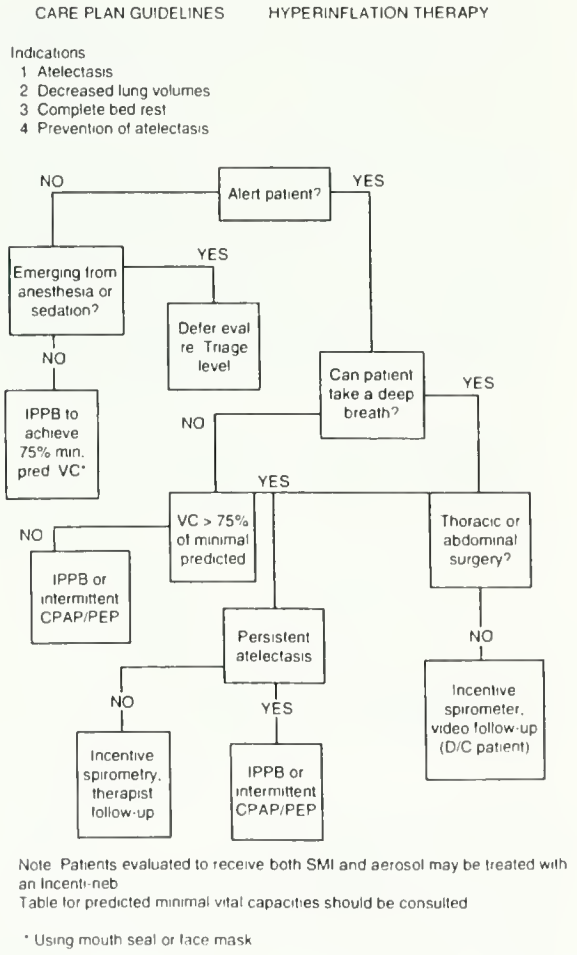
Mental status: obtunded, barely responsive
Level of activity: non-ambulatory
Chest x-ray: pleural effusion present in right hemithorax

B. Indicated Care Plan Therapies:

1. Oxygen therapy
2. Bronchopulmonary hygiene
3. Intermittent positive pressure breathing

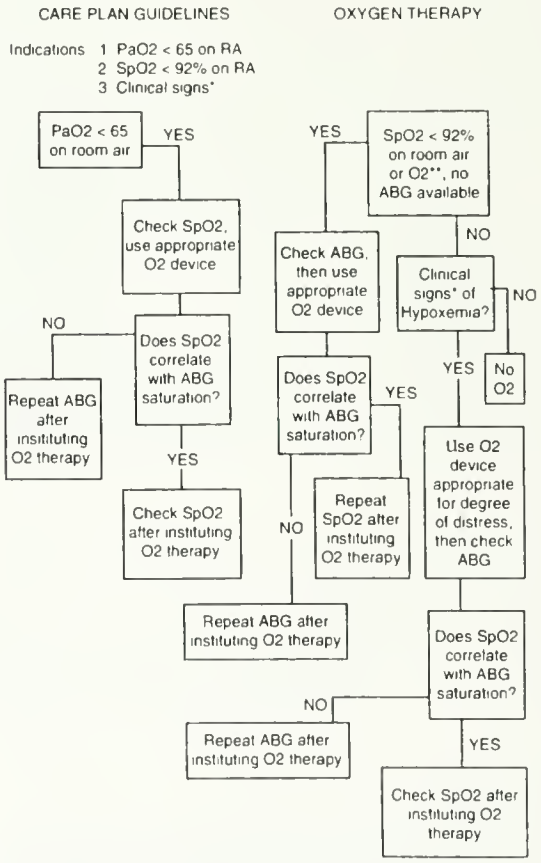
C. Relevant Algorithms for Case Study:





Note Patients evaluated to receive both SMI and aerosol may be treated with an Incenti-neb
Table for predicted minimal vital capacities should be consulted

* Using mouth seal or face mask


CARE PLAN GUIDELINES
OXYGEN THERAPY
Indications 1 PaO2 < 65 on RA
2 SpO2 < 92% on RA
3 Clinical signs*
PaO2 < 65 on room air
YES
Check SpO2, use appropriate O2 device
NO
Does SpO2 correlate with ABG saturation?
Repeat ABG after insitituting O2 therapy
YES
Check SpO2 after instituting O2 therapy
YES
SpO2 < 92% on room air or O2**, no ABG available
NO
Check ABG, then use appropriate O2 device
Clinical signs* of Hypoxemia?
NO
YES
No O2
Does SpO2 correlate with ABG saturation?
YES
NO
Repeat SpO2 after instituting O2 therapy
Use O2 device appropriate for degree of distress, then check ABG
Repeat ABG after instituting O2 therapy
Does SpO2 correlate with ABG saturation?
NO
Repeat ABG after instituting O2 therapy
YES
Check SpO2 after instituting O2 therapy

* SOB, tachycardia, diaphoresis, confusion, chest pain, cardiac history
** Patients already on O2 titrated as appropriate

Incidence of Ventilator-Associated Pneumonia (VAP) with 14-Day Circuit Change in a Subacute Environment

Robert E Thompson RPFT RRT

BACKGROUND: Studies suggest that decreased ventilator-circuit-change frequency results in decreased or unchanged incidence of VAP in acute care settings. However, few data have emerged from the subacute setting. This study compares the results of increasing the interval between ventilator circuit changes from 7 to 14 days in a subacute environment and also evaluates the incidence of nosocomial infection in patients with tracheostomies who were not receiving mechanical ventilation (MV). METHOD: During the first 6 months, tubing was changed every 7 days and during the second 6 months every 14 days for patients receiving MV ≥ 6 h/day. Once a patient achieved 24 consecutive hours of spontaneous breathing, that patient was placed into the tracheostomy group. Aerosol circuits for the tracheostomy group were changed 3 times/week throughout the study. Study patients were evaluated by clinical criteria for presence of leukocytosis, fever, purulent secretions, evidence of new infiltrate on chest film, and confirmation by the medical director. All patients with a length of stay < 5 days were excluded. RESULTS: The incidence of VAP with 7-day circuit changes was 3/31 versus 2/18 (p = 0.742) and the incidence of nosocomial pneumonia in the tracheostomy group was 4/22 for 7-day change intervals and 4/16 for 14-day intervals (p = 0.742). Aggregate incidence of pneumonia for ventilated patients (mean MV days = 55.4) was 10.2% and for the tracheostomy group 21%. CONCLUSIONS: 14-day circuit-change intervals result in an incidence of pneumonia that differs little from that seen with 7-day change intervals in this subacute environment. More data are needed to determine the maximum safe interval for circuit changes, especially in long-term patients in alternative care sites. [Respir Care 1996;41(7):601-606]

Background

Ventilator-associated pneumonia (VAP) refers to bacterial pneumonia developing in patients who have been receiving mechanical ventilation for at least 48 hours.[1] The acute care setting has been the site of a number of studies of the association between VAP and ventilator-circuit-change frequency. This study in a subacute environment is intended to add to existing data. Among common types of nosocomial infection, pneumonia is responsible for the greatest mortality. Incidence in mechanically ventilated patients ranges from 9 to 67%,[2-7] with mortality rates reported at ≥ 20% and sometimes as high as 76%.[8] Patients who develop nosocomial pneumonia have a 2- to 2.5-fold increased risk of death.[9] Many predisposing factors have been reported by authors regarding the development of VAP. These include the aspiration of bacteria colonizing the oropharynx or upper gastrointestinal tract,[10] gastric acid inhibition,[11] patient positioning,[12] contamination of respiratory care equipment,[12-14] frequent ventilator circuit changes (< 48 h),[5] and duration of mechanical ventilation.[2]

In the 1960s, increases in nosocomial pneumonia were found to be associated with the use of large-volume nebulizers

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for humidification.[15-17] Subsequent adoption of heated humidifiers appeared to reduce the likelihood of such infections. An important publication by Lareau et al[18] addressing ventilator tubing-change frequency and VAP appeared in 1978. This study demonstrated that circuit change intervals of 8, 16, and 24 hours resulted in no difference in VAP rates. Craven et al[19] in 1982 compared circuit contamination at 24 hours and 48 hours and found no difference in contamination levels. Their 1986 publication[20] demonstrated that the incidence of VAP was higher for 24-hour intervals than for 48-hour intervals. More recently, other studies have demonstrated reduction in VAP when circuits were changed less frequently than every 48 hours.[21-26] Two publications have demonstrated no change in the incidence of VAP with no routine circuit change.[7,20] The 1994 Center for Disease Control (CDC) Guideline[27] recommends that ventilator circuits be changed less frequently than every 48 hours. However, the maximum amount of time a ventilator circuit can remain in the system without significant change in VAP remains unknown. Although mounting evidence is encouraging, more investigation is needed particularly in subacute environments in which patients typically are ventilated for longer periods of time.

Because of the movement of the health care industry away from fee for service and toward managed care and capitated forms of reimbursement, cost has become an important issue for hospitals and subacute facilities. Recent estimates published by the CDC suggest that nosocomial pneumonia results in an increased length of stay (LOS) of 5.9 days and $5,683 in extra hospital charges for each episode (1992 dollars).[28] One study showed that no more than 5% of these excess costs would be recovered by hospitals under prospective payment.[29] Another study involving Medicare patients showed a median net loss of $5,800 per patient.[30] Clinical re-engineering and 'downsizing' of respiratory care departments raises a compelling challenge to reduce the cost of care and continue to provide safe and effective clinical management of mechanically ventilated patients.

Method

This study was conducted in the 25-bed ventilator unit of a 140-bed subacute facility located in a southwest suburb of Chicago, Illinois, during the 12-month period from October 1994 through September 1995. The ventilator unit was in its sixth year of operation at the time of study. During the first 6 months, all ventilated patients had their ventilator circuits* changed at 7-day intervals compared to 14-day intervals during the subsequent 6-month period. Spontaneously breathing patients with tracheostomies were also monitored throughout the 12-month period. All patients involved in the study were concurrently reviewed. Patients were not randomized in this study because only patients in the one unit were available for study and the sample size was small. Tracheostomy patients had aerosol tubing, drain bags, and tracheostomy masks changed 3 times per week throughout the study. All patients involved in the study received tracheostomy care every 12 hours by respiratory care practitioners. Prior to admission, patients were evaluated on-site at the referring facilities. Pre-admission evaluations were performed by the respiratory care manager and/or the nursing clinical coordinator of the ventilator unit. All patients admitted to the ventilator unit were assessed for pre-existing abnormal chest film and/or evidence of pneumonia as obtained from chart review by the clinical evaluators. The clinical criteria used to determine VAP were (1) change in secretion characteristics, (2) evidence of new infiltrate on chest film, (3) leukocytosis, (4) fever. In addition, the fact that each case met criteria was confirmed by the medical director of the unit, a board-certified pulmonologist.

Clinical data were collected during the course of each week. When a patient met criteria, consultation during weekly rounds with the medical director was sought to rule out other causes of infection. All data were collated at the end of each month. Patients were considered ventilator patients if they received 6 hours or more of mechanical ventilation per day. When patients were able to achieve 24 hours of consecutive spontaneous breathing, they were placed in the tracheostomy study group. If reinstitution of mechanical ventilation was required, the patient was returned to the ventilator study group. All patients with a LOS < 5 days were excluded from the study.

The presence of an established tracheal stoma is usually a pre-admission requirement for the unit. Those patients who were admitted to the unit without an artificial airway were not included in the calculation of nosocomial pneumonia data for this study. All patients were diagnostically classified based on a publication by Scheinhorn et al[31] (Table 1). Patients were also prognostically classified as to their rehabilitative or custodial status based on criteria in a publication by Cordasco[32] (Table 2).

Table 1. Classification of Subacute-Care Patients in Period 1 (7-Day-Change Interval) and Period 2 (14-Day-Change Interval) according to Schema of Scheinhorn et al[31]

Disease Classification	Ventilator Patients (%)		Tracheostomy Patients (%)	
	Period 1	Period 2	Period 1	Period 2
Chronic lung	32	28.5	14	6
Neurologic	26	26	68	75
Acute lung	13	17	4.5	0
Postoperative	16	28.5	4.5	6
Cardiac	13	18.5	4.5	18
Other	0	0	4.5	0

*Suppliers of commercial products are listed in the Product Sources Section at the end of the text.

Table 2. Characteristics, Number of Cases, and Incidence of Pneumonia in Subacute Care Patients in Period 1 (7-Day-Change Interval) and Period 2 (14-Day-Change Interval)

Variable	Ventilator Patients		Tracheostomy Patients	
	Period 1	Period 2	Period 1	Period 2
Number of patients (M/F)	31 (22/11)	18 (4/14)	22 (4/18)	16 (9/7)
Average age, years	70.2	65.6	59.2	54.1
Patient type, chronic/rehabilitation	17/16	8/10	18/4	14/2
Days of data collection	1538	1225	2001	2147
Cases of pneumonia detected	3	2*	4	4†
Number/1000 days	1.95	1.63	1.99	1.86

*$p = 0.742$ [χ^2 (1, 0.95)] for Period 1 (3/31) vs Period 2 (2/18); †$p = 0.916$ [χ^2 (1, 0.95)] for Period 1 (4/22) vs Period 2 (4/16).

Other than the reduction in the frequency of ventilator circuit change, no other changes in typical practice occurred. All staff gave careful attention to evaluating and identifying patients at risk for aspiration. Procedures such as elevating the patient's head, identification of aspiration by evaluation of swallowing (at the bedside) and by barium-swallow studies were common practices utilized at this facility. Histamine-receptor blockers were avoided, and sucralfate was typically administered for stress-ulcer prophylaxis. Aerosolized medication via small volume nebulizer was typically used throughout the course of this study.

χ^2 analysis was utilized to compare incidence of VAP with 7-day circuit change to incidence with 14-day circuit change.

Results

A total of 87 patients were included in the study, with 53 patients included during the first 6 months and 34 new admissions added during the second 6 months. Of those, 5 patients were dropped because their LOS was < 5 days, 49 patients were admitted to the ventilator unit as ventilator patients, and 38 were admitted as tracheostomy patients. Patients residing in the unit at the initiation of the study were included. The average number of days of mechanical ventilation was 55.4 days. Patient information, cases detected, rates/1000 days, and results of χ^2 analysis are shown in Table 2. Table 3 shows days of mechanical ventilation, days at detection of VAP, and cases detected overall.

During the course of the study, 21 patients were successfully weaned from mechanical ventilation and were placed in the tracheostomy study group. Two of these patients subsequently developed pneumonia and reinstitution of mechanical ventilation was required for hypoxic respiratory failure. Two other successfully weaned patients required reinstitution of mechanical ventilation for hypercapnic respiratory failure unrelated to pneumonia.

Table 3. Aggregate Statistics of Mechanically Ventilated Patients (MV) and Nonventilated Tracheostomy Patients (NVT)

Variable	MV Patients	NVT Patients
Mean days of MV	55	—
Mean MV days for patients developing VAP*	220.8	—
Days of MV before detection of VAP	129.3	—
Incidence of pneumonia	8/49	8/38
Number/1000 days	1.80	1.93

*VAP = ventilator-associated pneumonia.

Patients admitted to the subacute tracheostomy study group tended to be younger (57.0 y) and more custodial (84%) in nature, with a 71% prevalence of neurologic dysfunction. The ventilator group tended to be older (68.6 y) with more rehabilitative potential and a more even distribution of diagnoses than was present in the tracheostomy group.

Based on a review of the study group's pre-admission evaluations, 79.5% of the patients had abnormal chest radiographs prior to admission to the facility. These patients were referred by facilities including university, community, long-term, and rehabilitative hospitals representing many different physician groups and diagnostic methods. Because of this, there was no attempt to correlate a pre-admission pneumonia rate. One patient was admitted directly from home and was not included in determining the number of abnormal chest films prior to admission.

Discussion

Similar Studies

In a previously published abstract from the same subacute facility,[33] VAP rates were compared to four abstracts published in RESPIRATORY CARE[19,21-23] from acute care facilities using 7-day circuit change. The rates in the subacute study were lower than the average for acute care facilities (1.90 vs 6.08). Significance testing was not reported because it was believed that the comparison groups were dissimilar to our group of patients. Second, because 79.5% of the patients had abnormal chest radiographs prior to admission, it could be inferred that we might be looking at a second-time-pneumonia rate for this group of patients. Heated-wick wire circuits with non-reusable temperature probes were used throughout the study, which may have influenced the VAP rate. A recent abstract from New Zealand suggests less movement of contaminated liquids with heated-wire circuits.[34] A publication by Weems[13] identified a nosocomial outbreak of *Pseudomonas cepacia* related to reusable temperature probes. Decreased manipulation of the ventilator circuits may also have prevented unnecessary bolus aspiration of contaminated fluids into

patients' airways and may also have contributed to the lower rates seen with the 14-day circuit change group.

Limitations of Study

The study has several limitations important to interpretation of results. Diagnostic criteria may affect the reporting of VAP incidence. In this study, I utilized clinical criteria that have been previously shown to be less accurate than quantitative culturing methods employing protected-specimen-brush technique or bronchoalveolar lavage.[35,36] However, these procedures are not typical of subacute medical practice and were not available at the facility used for this study. The small sample size in the study precluded performing a controlled randomized study and increases the risk of a type-2, or β error. More than 14,000 patients would be required to detect the small differences observed in the incidence of VAP between groups.

Severity of illness may be an important factor explaining some of the difference between acute and subacute VAP rates because some degree of medical stability is required before admission to the subacute ventilator unit. Despite the limitations of using clinical criteria, the findings suggest the safety of decreased ventilator-circuit-change frequency in a subacute environment as has already been demonstrated by several acute care studies.

During the 2 phases of the study, clinical pneumonia developed in 10.2% of the mechanically ventilated group and 21% of tracheostomy study group. This lower percentage in mechanically ventilated patients may further support the commonly held belief that aspiration around artificial airways is the most likely culprit in the development of VAP.

Questions still remain regarding the maximum interval between ventilator tubing changes. Two randomized, controlled studies, Kollef et al[7] and Dreyfuss et al,[20] show little difference in VAP rates with no tubing change. However the average duration of mechanical ventilation for those studies was 15.7 and 11.6 days, respectively. Would similar results be found in patients who are receiving mechanical ventilation for more prolonged periods of time with no tubing change? In contrast, the average duration of mechanical ventilation in this study was 55.4 days (range 5-365, Fig. 1). The average number of days of mechanical ventilation received by patients who developed VAP was 220.8 days with an average detection time of 4.25 months after initiation of the study. A 1995 abstract by Fink et al[26] involving patients in respiratory and medical intensive care units revealed no difference between 7-day ventilator tubing changes and 30-day ventilator tubing changes. The average number of mechanically ventilated days was 11.75 days.

Implications & Projections

Based on the 1991 AARC-Gallup Poll survey,[37] it is estimated that approximately 11,419 mechanically ventilated patients are cared for in acute care hospitals in the continental U.S. at any given time. This population of patients undoubtedly continues to grow. Two combined studies of 998 patients showed that the average length of mechanical ventilation exceeded 7 days approximately 25% of the time.[38] The subacute-care industry has grown since the middle 1980s and throughout the 1990s, and it has been estimated that it will expand to a $10 billion industry by the end of the century.[39] Lower equipment and labor costs appear to be an obvious consequence of reducing ventilator tubing change frequency—a welcome reduction in the cost of care. In this study, based on an 8-ventilator/day average, the estimated cost savings from changing from 7- to 14-day circuit change was approximately $4,900 annually. In the future, more ventilated patients will probably reside in subacute facilities, a patient population that in past years would have been cared for in acute-care facilities. More studies are needed from subacute environments in which patients usually are ventilated for longer periods and in some instances indefinitely. Although the power of our study to detect a meaningful difference is low, these data provide a benchmark for future studies in patients who require long-term mechanical ventilation.

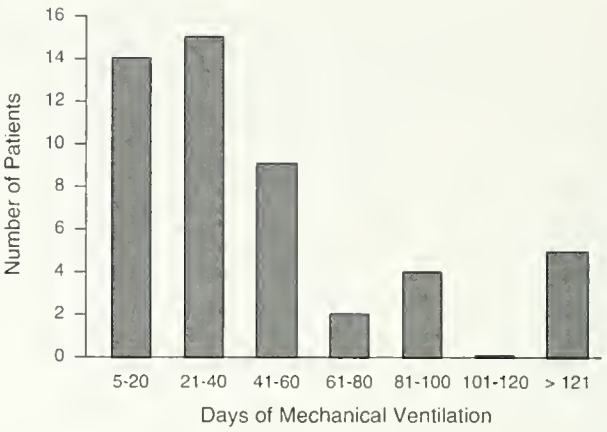

Fig. 1. Duration of mechanical ventilation for patients included in the study of ventilator-associated pneumonia with 7-day and 14-day circuit changes.

Conclusions

Similar results occurred in this subacute study with decreased ventilator-circuit-change frequency as has already been demonstrated in acute care settings. Increasing the interval of ventilator tubing change can be safely applied in subacute ventilator units. Lower VAP rates occurred in our study than in other studies performed in acute care environments. Health-care dollars can be saved, especially in facilities with large numbers of ventilator days. More information is needed from subacute facilities regarding VAP.

ACKNOWLEDGMENTS

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PRODUCT SOURCES

Ventilators:

Adult Star, Infrasonics Inc, San Diego CA
LP-6, Aequitron Medical Inc, Minneapolis MN

Tubing & Water Feed System for Ventilators:

AnaMed heated wick circuit (No. A8351 & A2668) & AnaMed Water Pump (No. A8000), Simplex Medical Systems Inc, Las Vegas NV
3000-mL water bags (No. 3175), IPI Medical Products, Chicago IL

Equipment for Tracheostomy Patients:

Misty-Ox Multi-Fit Nebulizers (No. 441A), Medical Molding Corp of America, Costa Mesa CA
Tracheostomy masks (No. 1075) & drainage bags (No. 1742), Hudson RCI Inc, Temecula CA
1000-mL water (No. 1065) & corrugated tubing (No. 3100), IPI Medical Products, Chicago IL

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CORRECTED FIGURE

The y-axis of Figure 17 in "Use of Inhaled Nitric Oxide in Patients with Acute Respiratory Distress Syndrome" by Hess D, Bigatello L, Kacmarek RM, Ritz R, Head CA, Hurford WE [Respir Care 1996;41(5):424-446], was incorrectly labeled ppm. The label should have been ppb (parts/billion). We regret the error.

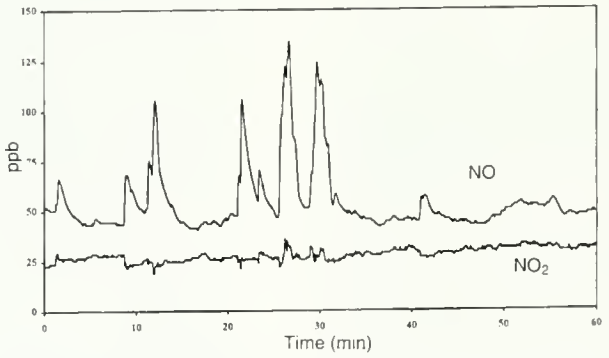

Fig. 17. Ambient NO and NO_2 levels for 1 hour with 100 ppm NO delivered into an ICU room at 8 L/min. NO and NO_2 measured by chemiluminescence 10 ft from NO flow. Note that ambient NO and NO_2 levels remain low with no scavenging other than usual room ventilation.

Evaluating the Oxygen-Tension-Based Indices of Venous Admixture: An Overview

Wesley M Granger PhD RRT

Introduction
$P_{(A-a)O_2}$ as an Index of $\dot{Q}_s/\dot{Q}_t$
P_{aO_2}/P_{AO_2} as an Index of $\dot{Q}_s/\dot{Q}_t$
P_{aO_2}/F_{IO_2} as an Index of $\dot{Q}_s/\dot{Q}_t$
Estimated Venous Admixture as an Index of $\dot{Q}_s/\dot{Q}_t$
Respiratory Index To Estimate $\dot{Q}_s/\dot{Q}_t$
Comparisons among O_2-Tension-Based Indices

[Respir Care 1996;41(7):607-610]

Introduction

Because the measurement and calculation of the physiologic shunt fraction, or venous admixture ($\dot{Q}_s/\dot{Q}_t$), involves an invasive procedure, clinicians have attempted to develop an accurate bedside method of estimating $\dot{Q}_s/\dot{Q}_t$. Elsewhere in this issue of RESPIRATORY CARE, I present a study of a computer model designed to estimate $\dot{Q}_s/\dot{Q}_t$ and P_{aO_2}.[1] In this piece, I review the work of the past 40 years on this topic—by way of introduction.

$P_{(A-a)O_2}$ as an Index of $\dot{Q}_s/\dot{Q}_t$

Two studies carried out in 1980 looked at the effects of varying F_{IO_2} on $\dot{Q}_s/\dot{Q}_t$.[2,3] These studies showed that as the F_{IO_2} is increased from 0.21 to 0.4, $\dot{Q}_s/\dot{Q}_t$ decreases and then remains constant as F_{IO_2} changes from 0.4 to 0.7. Then $\dot{Q}_s/\dot{Q}_t$ increases as F_{IO_2} is increased from 0.7 to 1.0. Based on these studies, there appears to be a physiologic effect of varying F_{IO_2} on $\dot{Q}_s/\dot{Q}_t$.

Investigators began to look for a mathematical index that could be used to noninvasively estimate $\dot{Q}_s/\dot{Q}_t$. The first of these indices was the difference in O_2 tensions between the alveoli and arterial blood ($P_{(A-a)O_2}$). This index became popular both because it is easy to calculate and because it is measured only while the patient breathes 100% O_2 and, therefore, the determination of $\dot{Q}_s/\dot{Q}_t$ is not affected by F_{IO_2}. When it became apparent that breathing 100% O_2 affected the measurement of true $\dot{Q}_s/\dot{Q}_t$,[2,3] clinicians began to measure shunt at whatever F_{IO_2} the patient was receiving. Under such conditions, the $P_{(A-a)O_2}$ is more variable and the relationship between venous admixture and $P_{(A-a)O_2}$ is much more complicated.

Keefer et al[4] sought to determine whether changes in $P_{(A-a)O_2}$ accurately reflect changes in $\dot{Q}_s/\dot{Q}_t$. They found that $P_{(A-a)O_2}$ and $\dot{Q}_s/\dot{Q}_t$ changed in the same direction in only 69% of their measurements and in opposite directions in 25% of the measurements. They concluded that using the change in $P_{(A-a)O_2}$ as an indicator of changes in $\dot{Q}_s/\dot{Q}_t$ is not reliable and should not be used, especially in critically ill patients. A study by Torda[5] in 1981 also looked at the relationship between $P_{(A-a)O_2}$ and $\dot{Q}_s/\dot{Q}_t$. This study showed that the effects of changes in cardiac output ($\dot{Q}_t$) and the difference in O_2 content between arterial and mixed-venous blood [$C_{(a-\bar{v})O_2}$] had a major affect on $P_{(A-a)O_2}$. The conclusion from the study was that $P_{(A-a)O_2}$ is not a reliable indicator of $\dot{Q}_s/\dot{Q}_t$. In a letter to the editor in 1987, Räsänen and Downs[6] discussed the available information on the use of $P_{(A-a)O_2}$ as an indicator of $\dot{Q}_s/\dot{Q}_t$. They concluded that $P_{(A-a)O_2}$ is not a reliable indicator of $\dot{Q}_s/\dot{Q}_t$, especially under conditions of changing F_{IO_2}. Therefore, it appears that $P_{(A-a)O_2}$ should not be used clinically as an index of gas-exchange impairment.

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P_{aO_2}/P_{AO_2} as an Index of $\dot{Q}_s/\dot{Q}_t$

The gas-exchange indices most often reported in the literature are the ratios of arterial-to-alveolar P_{O_2} (P_{aO_2}/P_{AO_2}) and of P_{aO_2} to F_{IO_2} (P_{aO_2}/F_{IO_2}). Gilbert and Keighley[7] were the first to study P_{aO_2}/P_{AO_2} under conditions of varying F_{IO_2}s. Their study consisted of three groups of patients. Group 1 included normal adults, Group 2 included 34 patients with pulmonary disease, and Group 3 consisted of 7 patients on mechanical ventilation. Groups 1 and 2 were given F_{IO_2}s of 0.21, 0.24, 0.28, 0.35, 0.40, and 1.0. Group 3 patients were studied at the F_{IO_2} set on the ventilator, which was randomly changed to 0.21, 0.3, 0.4, 0.6, and 1.0. Their results showed that the P_{aO_2}/P_{AO_2} was relatively constant with changes in F_{IO_2} compared to the $P_{(A-a)O_2}$ in Group 1. The P_{aO_2}/P_{AO_2} was seen to decrease slightly up to an F_{IO_2} of 0.4 and then increase slightly as F_{IO_2} increased to 1.0 in Group 2 and 3 patients. They concluded that the P_{aO_2}/P_{AO_2} was a better indicator of gas exchange with changes in F_{IO_2} because it was relatively stable over the range of concentrations studied. However, it can be shown using a mathematical model that if P_{aO_2}/P_{AO_2} is constant with changes in F_{IO_2}, then $\dot{Q}_s/\dot{Q}_t$ must be changing as F_{IO_2} changes.

See related study on Page 586.

Gilbert et al[8] looked at the stability of P_{aO_2}/P_{AO_2} in a 3-compartment computer model and in 10 patients with respiratory failure. They were able to show that $P_{(A-a)O_2}$ was affected more by changing F_{IO_2} than was P_{aO_2}/P_{AO_2}. They also differentiated two groups of patients based on the pattern of change in P_{aO_2}/P_{AO_2} with F_{IO_2}. The first group showed a constant ratio with changes in F_{IO_2}, whereas the second group showed a decreasing ratio for concentrations from 0.21 to 0.40 and then a large increase in the ratio for concentrations from 0.40 to 1.0. The pattern in Group 1 was shown to be expected from the 3-compartment model when shunt was the predominant gas-exchange defect. The pattern seen in Group 2 was consistent with conditions in which both $\dot{Q}_s/\dot{Q}_t$ and lung regions with a low ratio of alveolar ventilation ($\dot{V}_A$) to capillary perfusion ($\dot{Q}_c$), or $\dot{V}_A/\dot{Q}_c$, are present, and the low $\dot{V}_A/\dot{Q}_c$ abnormality predominates.

Because P_{aO_2}/P_{AO_2} is relatively stable in most patients as F_{IO_2} is varied, some studies looked at the usefulness of this ratio in predicting a new P_{aO_2} that could be expected with any given change in F_{IO_2}. Abizanda and Lopez[9] found a significant correlation ($r = 0.91$, $p < 0.005$) between measured P_{aO_2} and the predicted P_{aO_2} based on calculations using the baseline P_{aO_2}/P_{AO_2}. A graphic approach to this prediction of P_{aO_2} with changes in F_{IO_2} was developed by Gross and Israel[10] in 1981. Their graphic nomogram also takes P_{aCO_2} into account. They found that the actual P_{aO_2} differed from that derived from their nomogram by a mean (SD) difference of 2.6 (4.8) torr (range 0-10 torr). They reported that the nomogram had an overall sensitivity of 85% and concluded that reliable levels of F_{IO_2} can be chosen using this nomogram.

A study by Peris et al[11] in 1983 looked at the behavior of the P_{aO_2}/P_{AO_2} with changing F_{IO_2} and in predicting the resulting P_{aO_2} after a change in F_{IO_2}. They found that P_{aO_2}/P_{AO_2} did not change after varying the F_{IO_2} in their patient population. They observed that P_{aO_2}/P_{AO_2} was a more reliable predictive index in patients with a large degree of shunting ($P_{aO_2}/P_{AO_2} < 0.55$) than in those with a mild degree of shunting ($P_{aO_2}/P_{AO_2} \geq 0.55$).

Cohen et al[12] looked at the usefulness of P_{aO_2}/P_{AO_2} in the care of infants with respiratory distress. They found the ratio to be stable with changes in F_{IO_2} in these infants. They also calculated the percent change in $\dot{Q}_s/\dot{Q}_t$ and P_{aO_2}/P_{AO_2} relative to the change in inspired oxygen tension (P_{IO_2}). Their results showed that there was no significant difference between $\dot{Q}_s/\dot{Q}_t$ and P_{aO_2}/P_{AO_2} when expressed as percent changes ($t = 0.063$, $p \geq 0.05$).

Maxwell et al[13] looked at the use of P_{aO_2}/P_{AO_2} to predict the F_{IO_2} needed for a desired P_{aO_2} in mechanically ventilated patients. They found that in some patients the change in P_{aO_2}/P_{AO_2} was ≤ 0.05 (Group 1), whereas a second group of patients showed a change > 0.05 (Group 2), with changes in F_{IO_2}. The correlation between predicted F_{IO_2} and actual F_{IO_2} needed for Group 1 patients was 0.95, whereas for Group 2 patients it was 0.47. They concluded that for patients who are stable and the P_{aO_2}/P_{AO_2} change is < 0.05, the needed F_{IO_2} to obtain a desired P_{aO_2} can be accurately calculated using P_{aO_2}/P_{AO_2}. In less stable patients in whom the ratio change is ≥ 0.05, predictions are less accurate, and care should be exercised when making changes in F_{IO_2} based on these calculations.

Viale et al[14] developed a theoretical 2-compartment computer model to study the relationship between P_{aO_2}/P_{AO_2} and changes in F_{IO_2}. They concluded that results from previous studies showing that P_{aO_2}/P_{AO_2} remains constant with changes in F_{IO_2} may be due to $\dot{Q}_s/\dot{Q}_t$ changing with F_{IO_2} in the manner shown by Douglas et al.[3]

A study in 1986 by Doyle[15] looked at the relationship between P_{aO_2}/P_{AO_2} and $\dot{Q}_s/\dot{Q}_t$ at different levels of F_{IO_2}. This study showed that P_{aO_2}/P_{AO_2} varies with F_{IO_2} especially at high levels of $\dot{Q}_s/\dot{Q}_t$. In a letter to the editor, Gilbert[16] (with reply by Viale), reinforced the idea that P_{aO_2}/P_{AO_2} may remain constant with changes in F_{IO_2} while the actual $\dot{Q}_s/\dot{Q}_t$ is changing.

P_{aO_2}/F_{IO_2} as an Index of $\dot{Q}_s/\dot{Q}_t$

The ratio of P_{aO_2} to F_{IO_2} (P_{aO_2}/F_{IO_2}) was introduced by Horowitz et al[17] in 1974. The normal range for this ratio is considered to be 300-500—a value < 250 suggests a substantial gas-exchange impairment. The computational advantage of this gas-exchange index is that only P_{aO_2} and F_{IO_2} are required. Martyn et al[18] looked at factors influencing this ratio in burn patients. They found that estimates of $\dot{Q}_s/\dot{Q}_t$ from P_{aO_2}/F_{IO_2}

were useful only when $C_{(a-\bar{v})O_2}$ was 3-5 mL/dL. They also determined that changes in $\dot{Q}_t$, oxygen consumption ($\dot{V}_{O_2}$), and arterial oxygen content (C_{aO_2}) can all affect this index. In 35% of their patients, they found the $C_{(a-\bar{v})O_2}$ to be either > 5 mL/dL or < 3 mL/dL, and under these conditions P_{aO_2}/F_{IO_2} did not correctly reflect changes in $\dot{Q}_s/\dot{Q}_t$.

Wallfisch et al[19] looked at the $P_{(A-a)O_2}$ and P_{aO_2}/F_{IO_2} in patients with the acute respiratory distress syndrome (ARDS). They found that changes in P_{aO_2}/F_{IO_2} accurately reflected changes in $\dot{Q}_s/\dot{Q}_t$ only 57% of the time. They concluded that P_{aO_2}/F_{IO_2} was not reliable enough to adequately monitor respiratory failure and its treatment.

In 1989, Bone et al[20] looked at P_{aO_2}/F_{IO_2} as a predictor of survival in ARDS. The P_{aO_2}/F_{IO_2} was not significantly different at the time of ARDS diagnosis between those who ultimately survived and those who died. After the start of conventional therapy including positive end-expiratory pressure (PEEP), P_{aO_2}/F_{IO_2} was significantly different beginning on Day 1 of the study. Those patients who ultimately survived were able to increase their P_{aO_2}/F_{IO_2} in response to conventional therapy. The nonsurvivors were unable to improve their P_{aO_2}/F_{IO_2} even after 7 days of therapy. They concluded that the response of P_{aO_2}/F_{IO_2} to conventional therapy can be used to predict survival. This prediction aspect of the P_{aO_2}/F_{IO_2} may be its most useful clinical application in patients with ARDS.

Estimated Venous Admixture as an Index of $\dot{Q}_s/\dot{Q}_t$

Estimated venous admixture (EVA), or the estimated $\dot{Q}_s/\dot{Q}_t$, has been used as an index of gas exchange. This calculation involves assuming that $C_{(a-\bar{v})O_2}$ is constant at 5 mL/dL. However, studies in critically ill patients have shown that an assumed $C_{(a-\bar{v})O_2}$ of 3.5 mL/dL results in more accurate estimates of the true $\dot{Q}_s/\dot{Q}_t$.[21] A study by Hess et al[22] compared the classic shunt equation with the EVA using an assumed $C_{(a-\bar{v})O_2}$ of 3.5 mL/dL. They showed that the $\dot{Q}_s/\dot{Q}_t$ calculated from the classic equation may be from 6.3% greater to 3.3% less than the $\dot{Q}_s/\dot{Q}_t$ calculated from EVA equation. They concluded that EVA is a reasonable estimate of $\dot{Q}_s/\dot{Q}_t$ in hemodynamically stable patients. If the true $C_{(a-\bar{v})O_2}$ differs significantly from 3.5 mL/dL, then the error in the estimate increases, and it should not be used in patients who are not hemodynamically stable.

Respiratory Index To Estimate $\dot{Q}_s/\dot{Q}_t$

Respiratory Index (RI), $P_{(A-a)O_2}/P_{aO_2}$, was developed in 1975 by Goldfarb et al[23] as an index of respiratory status in trauma patients. In a group of 177 trauma patients, they established that normal values of RI are 0.1-0.37. Patients with RI ≥ 2 required intubation, and an RI ≥ 6 was associated with only a 12% chance of survival.

Hegyi and Hiatt[24] studied RI in infants with respiratory distress syndrome (RDS). They identified 3 groups of infants based on their RI: Group 1—RI = 0-3, Group 2—RI = 3-6, and Group 3—RI > 6. Continuous positive airway pressure (CPAP) was started at 6 cm H_2O, and the change in P_{aO_2} was noted: Group-1 P_{aO_2} increased by 23 torr, Group-2 P_{aO_2} increased by 5 torr, and Group-3 P_{aO_2} decreased by 10 torr. They concluded that RI may be useful in the initial assessment of disease severity for patients with RDS.

Comparisons among O_2-Tension-Based Indices

The O_2-tension-derived indices of gas exchange are: $P_{(A-a)O_2}$, P_{aO_2}/P_{AO_2}, P_{aO_2}/F_{IO_2}, and RI. The estimated venous admixture (EVA) is a combined O_2-tension- and oxyhemoglobin-saturation-based index because both P_{O_2} and S_{O_2} are required in the calculation. Covelli et al[25] studied all of the O_2-tension-derived indices as estimates of $\dot{Q}_s/\dot{Q}_t$ in 33 patients with ARDS. They calculated the correlation coefficients for each index against $\dot{Q}_s/\dot{Q}_t$. However, correlation is not the most appropriate statistical analysis for these data. Correlation shows only whether a relationship exists between two variables. How well one method estimates the true value of a variable is better assessed using the mean difference between the two variables (bias) and the standard deviation of this difference (precision, or limits of agreement).[26]

An editorial by Hess and Maxwell[27] in this Journal discussed the $P_{(A-a)O_2}$, P_{aO_2}/P_{AO_2}, and P_{aO_2}/F_{IO_2} in an attempt to determine which one was the best index for monitoring oxygenation. They concluded that P_{aO_2}/P_{AO_2} was the best of the three because it remains fairly constant with changes in F_{IO_2} and can be used to predict the F_{IO_2} needed to achieve a desired P_{aO_2}.

Dean et al[28] studied the O_2-tension-based indices as estimates of $\dot{Q}_s/\dot{Q}_t$ in critically ill children. They concluded that the O_2-tension-based indices do not accurately reflect $\dot{Q}_s/\dot{Q}_t$ in critically ill children. They also reported that when $C_{(a-\bar{v})O_2}$ was normal, arterial hemoglobin saturation (S_{aO_2}) became a very accurate assessment of $\dot{Q}_s/\dot{Q}_t$ (r = 0.96).

In 1988, Zetterström[29] studied all the O_2-tension-based indices as estimates of venous admixture. He also included P_{aO_2} and S_{aO_2} as possible estimates of $\dot{Q}_s/\dot{Q}_t$. Among these variables, P_{aO_2}/F_{IO_2} was the most accurate (r = 0.88) but was not independent of the F_{IO_2} used. The calculation of EVA using an assumed $C_{(a-\bar{v})O_2}$ of 5 mL/dL gave the best correlation with $\dot{Q}_s/\dot{Q}_t$ (r = 0.95). He concluded that when values of mixed-venous blood variables are not available, EVA is the best estimate of oxygenation status.

An identical study was reported by Cane et al[30] using 75 critically ill patients. They also found very poor correlations between the O_2-tension-based indices and $\dot{Q}_s/\dot{Q}_t$. The best correlation was found with the EVA using a $C_{(a-\bar{v})O_2}$ of 3.5 mL/dL (r = 0.94). Poor correlations were found for $P_{(A-a)O_2}$ (r = 0.62), P_{aO_2}/P_{AO_2} (r = 0.72), P_{aO_2}/F_{IO_2} (r = 0.71), and RI (r = 0.74). They concluded that the O_2-tension-based indices are unreliable indicators of $\dot{Q}_s/\dot{Q}_t$.

Herrick et al[31] studied how changes in the O_2-tension-based indices are reflected by changes in $\dot{Q}_s/\dot{Q}_t$. They found that none of the O_2-tension-based indices reliably reflect the behavior of $\dot{Q}_s/\dot{Q}_t$ with changes in F_{IO_2}. As the F_{IO_2} was changed from 0.3 to 1.0, 42% to 55% of the measured changes in these indices were opposite in direction to the changes in $\dot{Q}_s/\dot{Q}_t$. They concluded that the use of any of these indices for assessment of gas exchange when F_{IO_2} is changing can be misleading.

The computer model of gas exchange presented in this issue[1] is an attempt to mathematically evaluate the usefulness of the O_2-tension-based indices of gas exchange. This model shows that the most common bedside indices are not accurate predictors of true venous admixture. EVA, which uses both oxygen tensions and saturations, is the most stable of all these indices of gas exchange and more closely reflects true venous admixture.

The search continues for a noninvasive index of true venous admixture. However, one may not exist. Recent research has evaluated invasive indices that offer continuous monitoring of venous admixture. The shunt index (SI) and the ventilation-perfusion index (VQI) require the invasive measurement of pulmonary artery S_{O_2} but offer the advantage of continuous measurements.

Future research must focus on large populations to determine prediction equations based on the O_2-tension-based indices.

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The Effects of Flow Patterns on Pulmonary Gas Exchange, Lung-Thorax Mechanics, and Circulation

Richard Kallet MS RRT

Introduction
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Effects on Alveolar Residence Time for Gas Exchange
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Effects on Lung Mechanics
Effects on Peak Inspiratory Pressure
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[Respir Care 1996;41(7):611-624]

Introduction

Although the importance of the effects of inspiratory flow patterns on cardiopulmonary function has been debated since the studies of Watson in the early 1960s,[1,2] increased use of pressure-controlled (PCV) and pressure-supported ventilation (PSV) has rekindled interest in those effects.

In this overview, I compare and contrast what Chatburn et al[3] have called "the philosophically opposite approaches to assisted ventilation" by classifying them as flow-controlled ventilation with a constant inspiratory flow (ie, a rectangular-flow waveform, with step changes in flow possible) and pressure-controlled ventilation with a constant inspiratory pressure (ie, a decreasimg-ramp flow waveform). With this classification, I then treat flow-controlled ventilation using a decreasimg-ramp flow waveform as an approximation of pres-

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sure control. My objective is to answer the question, Do the waveforms generated during flow-controlled ventilation and pressure-controlled ventilation affect pulmonary gas exchange, lung-thorax mechanics, and cardiovascular function in different ways?

Before attempting to answer this question directly based on clinical and laboratory evidence, I review some of the theoretical constructs predicting how these flow patterns may affect cardiopulmonary function. Then, I examine the literature to determine whether theoretical benefits of either flow waveform have been demonstrated experimentally.

Theoretical Effects on Gas Exchange of Decreasing-Ramp & Constant-Flow Patterns

The theoretical advantages for gas exchange with flow of either pattern can be categorized as (1) the effects on the rate in rise of alveolar pressure (P_{alv}), and the associated rise in mean airway pressure ($\bar{P}_{aw}$); (2) the effects upon alveolar residence time for gas exchange; and (3) the effects on the intrapulmonary distribution of tidal ventilation. At any point during inspiration, the airway pressure (P_{aw}) reflects the force needed to overcome both the total resistance to gas flow (R) and the elastance (or reciprocal of compliance) of the lung-thorax (E_{LT}), as expressed by the simplified equation of motion for the respiratory system:[4]

$$P_{aw} = PR + PE_{LT}; \quad [1]$$

$$P_{aw} = (\dot{V}_I)(R) + (V_T/C_{LT\text{-}stat}), \quad [2]$$

where $\dot{V}_I$ = inspiratory flow, V_T = tidal volume, and $C_{LT\text{-}stat}$ = static lung-thorax compliance.

Effects on Alveolar Pressure Rise & Mean Airway Pressure

Although early lung modeling[5,6] was based on the premise that P_{alv} rose smoothly regardless of the pressure waveform applied at the mouth, those models did not account for the effect of chest-wall impedance on the build-up of P_{alv}. Baker and Hahn[7] incorporated chest-wall resistance into their electrically powered lung model and found that the sudden application of gas flow (as occurs with both decreasing-ramp and rectangular-flow waveforms) produced an instantaneous pressure step at the alveolar and pleural levels. The magnitude of this initial pressure step was related to the magnitude of the initial flow, the step rise was small (< 2 cm H_2O). Baker et al[8] repeated this study in humans and dogs and confirmed that an instantaneous pressure step occurred in vivo at the alveolar level (Fig. 1). The pressure step appears insignificant under the conditions tested. (The study was performed on normal dogs and human subjects undergoing thoracotomy for unspecified reasons, with a square wave that achieved a peak flow of 1 L/s.) However, the peak flows that can be achieved with PCV on some ventilators can reach 3 L/s, and in disease states marked by abnormal chest-wall resistance the actual pressure step conceivably could be much higher.

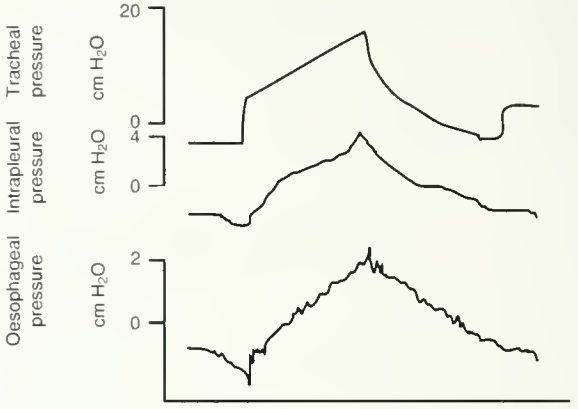

Fig. 1. Human tracheal, intrapleural, and esophageal pressure waveform responses to application of a constant-flow inspiratory pattern. Note that the sudden application of inspiratory flow causes an initial "pressure step" at all measurement sites. (Adapted from Reference 8, with permission)

Greater significance probably can be attributed to the rate of rise in alveolar pressure (ΔP_{alv}). From Equations 1 and 2 (simplified forms of the equation of motion), we can see that with constant inspiratory flow, the rate of rise in P_{alv} equals the $\dot{V}_I$ divided by $C_{LT\text{-}stat}$[5] as seen in Equation 3. Hence, for a given $C_{LT\text{-}stat}$, higher flows cause a more rapid rise in P_{alv} beyond the initial pressure step:

$$\Delta P_{alv} = \dot{V}_I/C_{LT\text{-}stat} = \frac{L/s}{L/cm\ H_2O} = cm\ H_2O/s. \quad [3]$$

However, for the constant inspiratory pressure waveform, alveolar pressure rises exponentially:

$$P_{alv}(t) = \Delta P_{aw}(1 - e^{-t/\tau}),$$
$$\text{and the initial rate of rise} = \Delta P_{aw}/\tau, \quad [4]$$

where ΔP_{aw} = peak inspiratory pressure – end-expiratory pressure, and τ = the time constant.

The difference in the rate of these waveforms may be easier to appreciate from Figure 2.[9]

The importance of these findings for PCV (or a decreasing-ramp flow pattern in volume-controlled ventilation, or VCV), apply to gas exchange problems seen in the acute respiratory distress syndrome (ARDS) and cardiogenic pulmonary edema. The logic behind the increase in oxygenation effected by the alveolar pressure step and the rapid pressure rise goes back to the work on hyaline membrane disease in the 1960s.

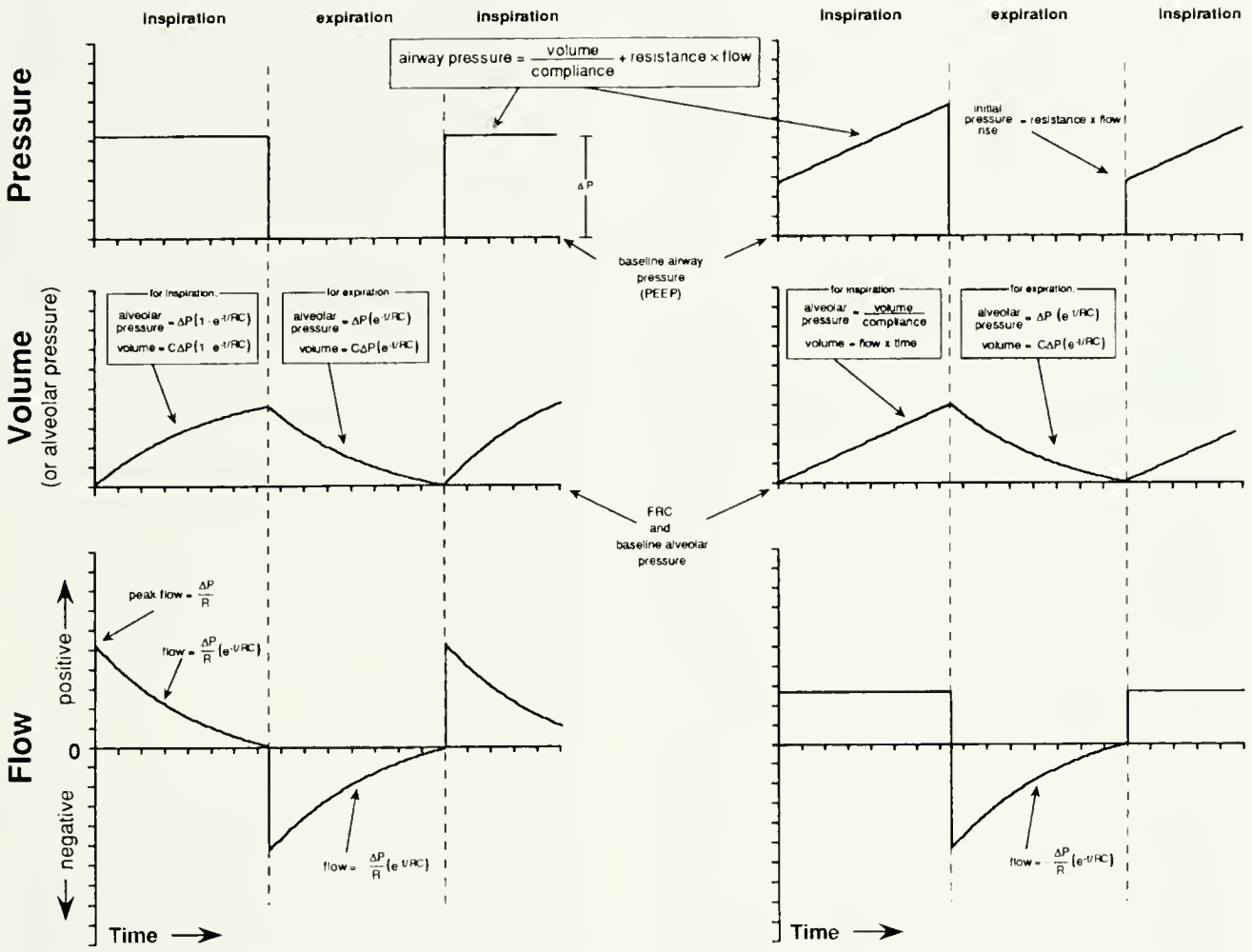

Fig. 2. Typical pressure, volume, and flow waveforms for pressure-controlled (rectangular-pressure waveform) and volume-controlled (rectangular-flow waveform) ventilation. Pressure, volume, and flow are shown as functions of time in accordance with the equation of motion (where muscle pressure = 0). Note that all variables are measured relative to their baseline, or end-expiratory values. ΔP = change in airway pressure; R = resistance; C = compliance; t = time; e = base of natural logarithm (≈ 2.72), FRC = functional residual capacity. (Reprinted from Reference 9, with permission)

Research on the mechanism of respiratory failure demonstrated that increased surface tension acting on alveoli leads to decreased compliance and to atelectasis.[10] Based on empirical evidence from other investigations, Reynolds[11] reasoned that collapsed alveoli reopen only when an inspiratory transpulmonary pressure threshold of 15-20 cm H_2O has been reached. This phenomenon was mentioned by Cherniack et al,[12] who observed that beyond the threshold of 20 cm H_2O, lung tissue recruitment continues unevenly until complete lung inflation has been achieved at approximately 40 cm H_2O. According to Reynolds, "It was therefore reasoned that if the alveoli could be held open for a longer proportion of each breath, oxygen uptake might be improved."[11]

The inspiratory pressure threshold to open collapsed alveoli has been described as a transpulmonary pressure-volume hysteresis or an inflection point (P-flex) on the inspiratory pressure-volume curve.[13] It has been shown that approximately 31% of the pulmonary units in early ARDS are collapsed *and* capable of being recruited with positive end-expiratory pressure (PEEP).[14] Furthermore, optimal oxygenation appears to require a set PEEP level greater than the P-flex point.[15] By extension, in disease states marked by diffuse alveolar collapse, the ability to create both a relatively higher alveolar pressure step and a more rapid ΔP_{alv} early in the inspiratory phase decreases the time needed to recruit alveoli. This would extend the inflation time of unstable alveoli and, thus, could improve oxygenation.

At equivalent tidal volumes and inspiratory times, a decreasing-ramp profile results in a higher $\bar{P}_{aw}$ and P_{alv} than does a rectangular-flow pattern. This derives from the instantaneous pressure rise and the fact that the majority of volume change occurs earlier in the inspiratory phase.[4,16] Ran and Shelledy[17] reported consistently higher $\bar{P}_{aw}$ with a decreasing-ramp waveform under both normal and high resistive and elastic con-

ditions in a mechanical lung-model study. Although the absolute differences in mean pressures were only 0.8-1.6 cm H_2O, the relative increase was approximately 15-20% with the decreasing-ramp waveform.[17] Increased $\overline{P}_{aw}$ has been associated with increased arterial oxygenation regardless of whether it is achieved by PEEP, duration of positive pressure, or airway pressure waveform.[18]

Effects on Alveolar Residence Time for Gas Exchange

Because the inspiratory flow dictates the rate of lung-volume change, the higher initial flow in a decreasing-ramp profile causes a more rapid delivery of V_T.[5] This, in turn, prolongs alveolar residence time for gas exchange and interalveolar gas mixing, and may also promote alveolar recruitment.[19,20] This can be seen in a computer lung-model study by Chatburn et al[3] in the time course for lung inflation between constant-pressure (ie, PCV) and constant-flow ventilation. Whereas lung-volume change was linear and reached its peak volume at the end of inspiration with constant flow, under constant-pressure ventilation the major portion of the V_T appears to have been delivered during the first 25% of inspiration.

Effects on Distribution of Inspired Tidal Ventilation

In a 1972 computer lung-model study, Jansson and Jonson[21] found that when the compliance, resistance, and time constants of two lung units were uniform, no difference was seen in the relative distribution of ventilation with either waveform. However, differences appeared when disturbances in impedance and time constants were unilaterally distributed—a finding confirmed by Chatburn et al.[3] These findings were consistent with those of Otis et al,[6] who found that tidal gas redistributes from the lung unit that reaches early equilibration to the slower filling unit. Chatburn et al found that the decreasing-ramp pattern effected more even distribution when airways resistance was unilaterally increased. When one lung unit had decreased compliance, more even distribution was achieved with a rectangular flow, whereas the decreasing ramp caused a larger portion of tidal ventilation to be diverted to the normal lung. Hyperinflation of normal lung tissue could cause a so-called reverse mismatch of ventilation and perfusion, similar to what has been reported when PEEP is applied in unilateral lung disease.[22,23]

Clinical studies of lung mechanics have shown that as inspiratory flows increase, resistance to inflation (attributed to the viscoelastic properties of the airways, lung, and chest wall) decreases.[24,25] The rapid rise in airway pressure, associated with high inspiratory flow, is thought to cause mechanical airway dilatation and decreased tissue resistance.[24] A higher pressure step and a more rapid rise in airway pressure may improve conductance and tidal volume distribution, by dilating and reopening narrowed and collapsed peripheral airways. Reopening collapsed airways early in the inspiratory phase may lessen the ventilation-perfusion mismatching seen in patients after upper abdominal surgery.[26] Given the similar increases in closing volume seen in cirrhotic liver and obstructive lung diseases,[27] the decreasing-ramp flow also may improve gas exchange in these situations. In addition, when regional airways resistance is not uniform, the spectrum of inspiratory flows generated during flow tapering may match regional resistances better than does a fixed flow.

To summarize, differences in gas exchange that occur with decreasing-ramp and constant-flow patterns may be associated with different mechanical disturbances. When the lungs are normal, the flow pattern should not affect gas exchange. When the underlying pathologic disturbance is obstructive, a decreasing-ramp pattern may effect better gas exchange by better distribution and earlier equilibration time. When diffuse, bilateral congestive atelectasis is present, a decreasing-ramp pattern may promote alveolar recruitment and improve gas exchange due to a more rapid ΔP_{alv}, longer inflation time, and, thus, a higher $\overline{P}_{aw}$. By contrast, when compliance is lower and more unilaterally distributed, a constant inspiratory flow may effect better gas exchange due to more even distribution of tidal volume.

Effects on Lung Mechanics

The relative effects of flow patterns on pulmonary mechanics can be predicted, in part, from the simplified version of the equation of motion for the pulmonary system (Equations 1 & 2).[4] The pressure required to overcome resistance is a function of both the inspiratory flow and resistance, and the pressure needed to overcome elastance is a function of displacement (tidal volume) and the elastic properties of the lungs and chest wall.[17] Therefore, the maximum pressure associated with resistance occurs at the highest flow, while the maximum pressure needed to overcome the elastic properties of the thorax occurs at the end of the breath, when the entire tidal volume has been delivered.[17]

Effects on Peak Inspiratory Pressure

In the rectangular pattern, the constant flow results in a constant pressure to overcome resistance throughout inspiration. At the same time, the linear flow (or rate of lung-volume change) results in a steady rise in pressure associated with elastic forces. Therefore, the peak inspiratory pressure (PIP) measured at the end of inspiration reflects the constant-flow resistive pressure plus the total elastic pressure.

In the decreasing-ramp pattern, flow is highest at the beginning of inspiration and continually decays throughout the inspiratory phase. As a consequence, the high initial pressure step that accompanies this pattern is a reflection of the resistance to higher peak flows. The early delivery of tidal volume causes a rapid rise in elastic pressure that steadily replaces the resistive pressure as flow tapers. PIP measured at the end of inspi-

ration primarily reflects the elastic pressure because flow either tapers to a low value or has ceased. This depends on the mode of ventilation. In VCV with a decreasing-ramp pattern, the flow tapering is predetermined. In PCV, the flow tapering is determined entirely by the lung mechanics. Therefore, in states characterized by low resistance and low compliance, flow may have reached zero well before the end of the inspiratory phase. Under conditions of high resistance and normal or high compliance, inspiratory flow may be high at the end of inspiration. Therefore, PIP reflects both resistive and elastic pressure. Accordingly, the PIP should be lower with decreasing-ramp flow. However, Rau and Shelledy[17] observed that PIP actually was higher with an exponentially decreasing-ramp pattern under conditions of high airway resistance. In this situation, the associated pressure waveform probably deviated from a rectangular pattern, and the PIP at the beginning of inspiration reflected an 'overshoot.'[28]

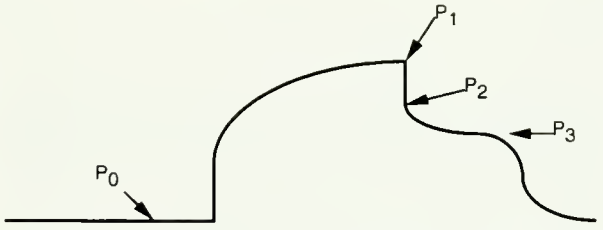

Fig. 3. Airway pressure contour with a constant flow pattern. P_0 = the end-expiratory pressure, P_1 = peak inspiratory airway pressure, P_2 = the pressure associated with both tissue resistance, uneven distribution of ventilation, and elastic recoil forces of the lung-thorax, P_3 = pressure needed to counter the elastic recoil forces of the lung-thorax. $P_1 - P_0$ = total dynamic compliance. $P_1 - P_2$ = pressure drop attributed to gas flow resistance. $P_2 - P_3$ represents the pressure drop associated with tissue viscoelastic resistance, and also, with the redistribution mechanical stress with end-inspiratory volume shifts (inspiratory pendelluft).

Effects on Dynamic Compliance & Flow Resistance

If PIP is lower with decreasing-ramp flow, then the dynamic compliance (C_{dyn}) should be higher. Dynamic compliance, or "dynamic characteristic" is an index reflecting the inflation of the thorax with a specific volume of gas, relative to the force exerted by the ventilator to achieve it [$V_T/(PIP - PEEP)$]. However, caution should be exercised when interpreting the significance of an improvement in C_{dyn} because measurement accounts not only for the applied force necessary to overcome the elastic properties of the lung and thorax but also for the total resistive properties of the patient-ventilator circuit and the additional counter-elastic forces created by the presence of intrinsic PEEP.[29] Total flow resistance includes the frictional resistance and turbulence created during gas flow through the airways, plus, the viscoelastic resistance or stress adaptation caused by tissue deformation.[30-32] Viscoelastic resistance represents the friction developed within the tissue beds of the airways, lungs, and chest wall during dynamic inflation.[31] Resistance is differentiated by a two-phase pressure drop during an end-inspiratory pause (Fig. 3). The initial drop in pressure ($P_1 - P_2$, in Figure 3) represents the force required to overcome frictional resistance to gas flow.[32] The secondary pressure drop ($P_2 - P_3$) reflects both the force necessary to deform pulmonary and chest-wall tissue[32] and the mechanical unevenness in the distribution of stress associated with maldistribution of volume.[25,33]

An increase in C_{dyn} associated with a decreasing-ramp pattern may only represent a reapportionment of gas flow resistance (primarily from a rigid artificial airway) to the beginning of inspiration. To demonstrate an improvement in a patient's pulmonary mechanics with a decreasing-ramp pattern, requires measurement of the pressure drop associated with viscoelastic resistance and inspiratory pendelluft ($P_2 - P_3$, in Figure 3). A smaller pressure drop between P_2 and P_3 would reflect less mechanical unevenness within the lungs or less tissue resistance. For completeness it should be noted that the $V_T/(P_2 - P_0)$ is referred to as kinetic compliance (C_k) and reflects the summation of the elastic and viscoelastic properties of the lung-thorax.[34]

Based upon evidence presented in the previous section, a decreasing-ramp flow would be expected to improve tissue resistance because of the mechanical bronchodilation and early redistribution of tidal ventilation. In addition, the combined effects of decreased tissue resistance and flow tapering may also improve tidal distribution, and, therefore, lessen the unevenness of mechanical stress. This may be the case in primary obstructive diseases and, possibly, in ARDS. As noted earlier, when compliance is low and inhomogeneous in its distribution, flow in a rectangular pattern may effect better distribution of tidal ventilation and less tissue resistance.

Effects on Static Lung-Thorax Compliance

Static lung-thorax compliance ($C_{LT\text{-}stat}$) is an index reflecting the elastic recoil properties of the lung-thorax. It is determined by measuring the applied pressure necessary to hold a delivered V_T inside the chest. Therefore, it is measured under static conditions, and the relevant distending pressure is the difference between the equilibrated, end-inspiratory pressure plateau (EIP) that is achieved at zero flow and the end-expiratory alveolar pressure [$V_T/(EIP - EEP_{alv})$]. Changes in C_{dyn} are not specific and could reflect changes in either frictional resistance, tissue viscous resistance, elastic forces, or intrinsic PEEP. Recruitment of collapsed alveoli cannot be inferred from improvements in C_{dyn}, but rather from improvements in $C_{LT\text{-}stat}$. However, when disease is inhomogeneous or unilateral, any effects of lung-unit recruitment on static compliance may be obscured by concurrent overdistention of normal lung units, caused by maldistribution of tidal volume. In this case, a rectangular pattern may effect a more even distribution of tidal ventilation and a higher $C_{LT\text{-}stat}$.

Effects on Cardiovascular Function

Predicting the effects of flow patterns upon cardiovascular function is difficult. Cardiovascular performance during positive pressure ventilation depends on several factors, including the proportion of airway pressure transmitted to the pleural space (which depends upon lung and chest-wall compliances),[35] intravascular volume status, and vascular tone.[20] Any differences in the effects on cardiovascular function of flows of the two configurations is a function of differences in $\overline{P}_{aw}$. As mentioned earlier, $\overline{P}_{aw}$ is higher with a decreasing-ramp pattern. The absolute differences in mean pressures reported by Rau et al[17] were only 0.8-1.6 cm H_2O. Therefore, it seems unlikely that the inspiratory flow contour affects cardiovascular performance under most circumstances. However, cardiac output may be adversely affected by the decreasing-ramp profile (when airway pressure is transmitted readily to the pleural space) in the presence of poor vascular tone, low intravascular volume, or impaired right ventricular contractility.

Literature Review

Review Methods

A MEDLINE title-word search was conducted for the years 1966-1995, and a subject-heading search for the years 1960-1965 using *Index Medicus*. Three delimitations were imposed. First, in those articles in which manipulations of both end-inspiratory pause and inspiratory time (and inspiratory flow pattern) occurred, I used only data comparing constant and decreasing-ramp patterns. Therefore, in this review I do not consider experiments that compared inverse-ratio ventilation in either the volume-controlled or pressure-controlled modes. Second, I included experiments that incorporated fixed end-inspiratory pause into the study of waveforms in the analysis. Third, I excluded case studies and the pediatric literature.

Data Manipulation

Comparisons of data were expressed as a percentage of change between mean values obtained with a constant or decreasing-ramp waveform. Numeric comparisons were made only when the differences in values were reported by the investigators to have achieved statistical significance.

Results

In total, 16 articles met the specified criteria and were included in the review. A description of each study and some of the controls employed in the design are presented in Table 1. Comparisons of some of the flow patterns used in the reviewed studies can be found in Figure 4A-E. Table 2 lists the major dependent variables measured for each study. Of 13 studies in which gas exchange variables were measured, 6 (46%) revealed

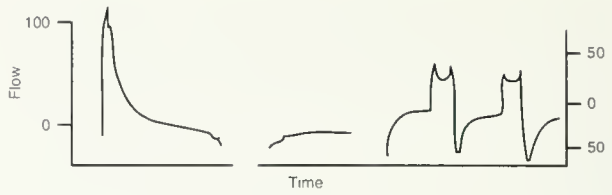

Fig. 4A. Decreasing-ramp and constant-flow inspiratory waveforms produced by Watson's (pressure generator) ventilator. (Adapted from Reference 46, with permission)

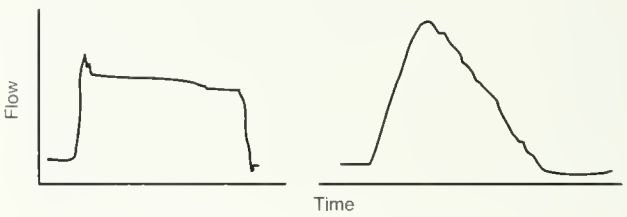

Fig. 4B. Constant-flow and decreasing-ramp waveforms produced by Adams et al's ventilator. (Reprinted from Reference 45, with permission).

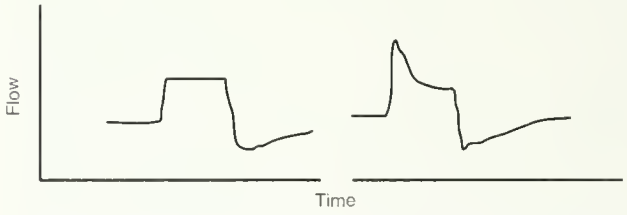

Fig. 4C. Constant-flow and decreasing-ramp inspiratory waveforms produced with a Siemens 900 ventilator used by Johansson and Lofstrom. (Adapted from Reference 47, with permission)

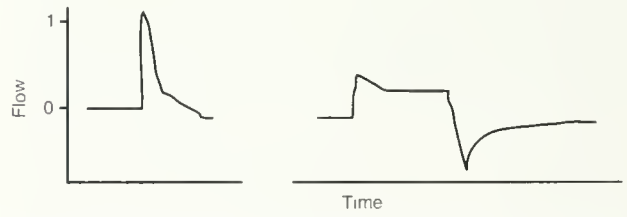

Fig. 4D. Decelerating and constant-flow inspiratory waveforms produced from Baker et al's experimental ventilator. (Adapted from Reference 41, with permission)

Fig. 4E. Constant-flow and decreasing-ramp waveforms produced by the BOC, Medishield IMV Pneumotron used by Al-Saady and Bennett. (Adapted from Reference 35, with permission).

Table 1. Experiments Examining the Effects of Constant and Decreasing-Ramp Flow Waveforms

First Author, Reference, Year	Ventilator(s) Used	Sample Type; Size (n)	Study Controls
Mang,[16] 1995	Siemens Servo 900C	Acute lung-injury model (sheep), n = 20	f, V_T, I:E, total PEEP, & F_{IO_2} held constant; random presentation of flow waveforms
Lessard,[36] 1994	Siemens Servo 900C	Medical ICU population, ARDS Score > 2.5, n = 9; both early & late ARDS	V_T, $\dot{V}_E$, T_I, & total PEEP held constant; 10% end-inspiratory pause used; random presentation of waveforms
Rappaport,[37] 1994	Siemens Servo 900C	Medical ICU population; $P_{aO_2}/F_{IO_2} < 150$, primarily pneumonia & sepsis (excluded COPD & asthma), n = 27	V_T, $\dot{V}_E$, & T_I/T_{TOT} held constant; 10% end-inspiratory pause; patients triggering ventilator during measurements; random assignment to treatment
Abraham,[38] 1990	Siemens Servo 900C	Medical-surgical ICU population; $P_{aO_2}/F_{IO_2} < 100$, n = 10	V_T, f, T_I, auto-PEEP, & F_{IO_2} held constant; presentations one-way: VC to PC
Smith,[39] 1988	Hamilton Veolar	Domestic swine with metacholine-induced bronchospasm, n = 7	V_T, f, & T_I held constant; random presentation of waveforms
Al-Saady,[34] 1985	BOC, Medishield IMV Pneumotron	Medical ICU population ARDS, pneumonia, acute-on-chronic failure, n = 14	V_T, $\dot{V}_E$, & I:E held constant; nonrandom presentation, repeated measures design
Baker,[40] 1982	Experimental ventilator controlling V_T, T_I, and waveform (Baker, 1971)	Papin-induced panlobar emphysema in mongrel dogs, n = 15	T_I & V_T constant; nonrandom presentation
Baker,[40] 1982	Experimental ventilator controlling V_T, T_I & waveform (Baker, 1971)	Oleic acid-induced ARDS in greyhound dogs, n = 15	T_I & V_T constant; nonrandom presentation
Baker,[41] 1977	Experimental ventilator controlling V_T, T_I, & waveform (Baker, 1971)	Normal mongrel dogs, n = 8	T_I & V_T constant; nonrandom presentation
Johansson,[42] 1975	Siemens Servo 900	Normal adults undergoing general anesthesia, n = 10	T_I, f constant, $\dot{V}_E$ not controlled (set to keep $P_{aCO_2} \approx 40$ torr), V_T differences not significant; waveforms randomly presented
Johansson,[42] 1975	Siemens Servo 900	Adult ICU patients with chronic bronchitis, sepsis, pneumonia, n = 5	f varied from 12-24; no control of T_I; waveforms randomly presented
Johansson,[43] 1975 Circulation study	Siemens Servo 900	Mix of adult ICU patients & patients under general anesthesia, n = 23	Only f controlled; waveforms randomly presented; part retrospective, part prospective study
Johansson,[44] 1975 Thoracic compliance	Siemens Servo 900	Mixture of adult ICU & patients under general anesthesia, n = 35	No control of ventilator settings during study; waveforms randomly presented
Adams,[45] 1970	Experimental ventilator	Normal dogs with normovolemia & induced hypo- and hypervolemia, n = 7	V_T, f, T_I held constant, $F_{IO_2} = 0.21$; 3/4 waveforms randomly presented
Watson,[46] 1962	Experimental ventilator	Studies of adults with neuromuscular disease, n = 3 & 5	V_T, f, & T_I controlled; end-inspiratory pause during measurements; random presentation of waveforms

T_I = inspiratory time, T_I/T_{TOT} = inspiratory time fraction, or duty cycle, VC = volume-controlled ventilation, $\dot{V}_E$ = minute ventilation, f = frequency of ventilator breath.

Table 2: Studies Comparing Effects of Decelerating and Constant Flow Waveforms on Gas Exchange, Mechanics, and Cardiorespiratory Variables

First Author, Reference, Year Patient Type, & n	Variables		
	Gas Exchange	Lung Mechanics	Circulatory & Cardiorespiratory
Mang,[16] 1995	P_{aO_2} NS; P_{aCO_2} NS; pH NS; $\dot{Q}_s/\dot{Q}_t$ NS; efficiency of alveolar ventilation NS	C_{dyn} NS	CO NS
Lessard,[36] 1994, ARDS; n=9	P_{aO_2} NS; $\dot{Q}_s/\dot{Q}_t$ NS; P_{aCO_2} NS; V_D/V_T NS	PIP: VC 13% > PC; $\bar{P}_{aw}$ NS; EIP NS; $C_{LT\text{-}stat}$ NS	CI NS; $\bar{P}_{art}$ NS; $\bar{P}_{pa}$ NS; P_{pcw} NS; $P_{(a\text{-}v)O_2}$ NS
Rappaport,[37] 1994, ARDS, n=27	P_{aO_2} NS; $\dot{Q}_s/\dot{Q}_t$ NS; P_{aO_2}/F_{IO_2} NS; P_{aCO_2} NS	$\bar{P}_{aw}$ NS; EIP NS; $C_{LT\text{-}stat}$ NS; R_{aw}: PC 30% < VC in nonsurvivors	—
Abraham,[34] 1990, ARDS; n=10	P_{aO_2}, PC 15%> VC; $\dot{Q}_s/\dot{Q}_t$ NS; P_{aCO_2} NS	$\bar{P}_{aw}$: NS; C_{dyn}: PC 35% > VC; PIP: VC 37.8% > PC	$\bar{P}_{art}$ NS; CI NS; $P_{(a\text{-}v)O_2}$ NS; P_{pcw} NS; P_{cv} NS; D_{O_2}: PC 9.8% > VC
Smith,[39] 1988, induced asthma, porcine model, n = 7	P_{aO_2} NS; P_{aCO_2} NS; VD/VT NS	P_{tr} NS	CO NS; $\bar{P}_{art}$ NS
Al-Saady,[34] 1985, mix of ICU patients, n = 14	P_{aO_2}: DF 14% > CF; $P_{(A\text{-}a)O_2}$: DF 4% < CF; P_{aCO_2} NS; V_D/V_T: DF 16% < CF	PIP: CF 16% > DF; EIP: 8% < CF; $C_{LT\text{-}stat}$: DF 17% > CF; C_{LTk}: DF 15% > CF; $\bar{P}_{aw}$ NS	CO NS; $\bar{P}_{art}$ NS; P_{pa} NS; P_{pcw} NS; P_{cv} NS
Baker,[49] 1982, pulmonary edema, dog model, n = 15	P_{aO_2} NS; $P_{(A\text{-}a)O_2}$ NS $\dot{Q}_s/\dot{Q}_t$ NS; P_{aCO_2}: DF 6% < CF; V_D/V_T NS	$\bar{P}_{aw}$: DF 20% > CF; $\bar{P}es$: DF 8% > CF	—
Baker,[40] 1982, emphysema, dog model; n = 15	P_{aO_2} NS; $P(_{A\text{-}a})O_2$: DF 9.6% > CF; $\dot{Q}_s/\dot{Q}_t$ NS; P_{aCO_2}: DF 5% < CF; V_D/V_T: DF 5% < CF	$\bar{P}_{aw}$: DF 29% > CF; $\bar{P}_{es}$: DF 13% > CF	$\bar{P}_{art}$ NS; HR NS; $\bar{P}_{pa}$ NS
Baker,[41] 1977, normal dogs; n = 8	P_{aO_2}: DF 7% > CF; $P(_{A\text{-}a})O_2$: DF 19% < CF; $\dot{Q}_s/\dot{Q}_t$ NS; P_{aCO_2}: DF 9% < CF; V_D/V_T: DF 12% < CF	C_{dyn}: DF 6% > CF; C_{cw}: DF 6% > CF; C_L NS; $\bar{P}_{aw}$: DF 30% > CF; $\bar{P}_{es}$: DF 20% > CF	CO NS
Johansson,[42] 1975; anesthesia; n = 10	P_{aO_2} NS; $P(_{A\text{-}a})O_2$: DF 24% < CF; P_{aCO_2}: DF 9% > CF; V_D/V_T DF 5% > CF	C_{dyn} NS; P_{tr} NS	—
Johansson,[42] 1975; ICU; n = 5 (gas exchange; n = 9 mechanics)	P_{aO_2} NS; P_{aCO_2} NS; V_D/V_T NS	C_{dyn} NS; P_{tr} NS	—
Johansson,[44] 1975, ICU & anesthesia; n = 35	—	C_{dyn} NS; C_{cw} NS; C_L NS	—
Johansson,[43] 1975; retrospective (anesthesia, n = 15) & prospective (ICU, n = 8)	—	—	HR NS; $\bar{P}_{pa}$ NS; P_{pcw} NS; P_{cv} NS; $a\text{-}vD_{O_2}$ NS
Adams,[45] 1970; normal dogs; n = 13	P_{aO_2} NS; $P_{(A\text{-}a)O_2}$ NS; $\dot{Q}_s/\dot{Q}_t$ NS; P_{aCO_2} NS; V_D/V_T NS; V_D NS	$\bar{P}_{aw}$ NS	$\bar{P}_{art}$ NS; CO NS; $P_{(a\text{-}v)O_2}$ NS
Watson,[46] 1962; paralyzed patients; n = 3	V_D/V_T NS	—	—
Watson,[46] 1962; paralyzed patients; n = 5	—	C_{dyn} NS	—

$\bar{P}_{art}$ = mean arterial pressure, C_{dyn} = dynamic compliance, CF = constant-flow waveform, $C_{LT\text{-}stat}$ = static lung-thorax compliance, C_{LTk} = kinetic lung-thorax compliance, DF = decreasing-ramp flow, D_{O_2} = systemic O_2 delivery or transport, EIP = end-inspiratory (plateau) pressure, n = number in study, NS = statistically nonsignificant difference, $P_{(A\text{-}a)O_2}$ = alveolar-arterial partial pressure difference, P_{aw} = airway pressure, PC = pressure-controlled ventilation, P_{cv} = central venous pressure, P_{es} = esophageal pressure, PIP = peak inspiratory (airway) pressure, P_{pa} = pulmonary artery pressure, P_{pcw} = pulmonary capillary wedge pressure, P_{tr} = tracheal pressure, R_{aw} = inspiratory airways resistance, VC = volume-controlled ventilation.

statistically significant differences. If an arbitrary point of 10% difference in values is set as the threshold for clinically important findings, then only 4 studies (31%) appear to have therapeutic consequences (Johansson and Lofstrom;[47] Baker et al;[41] Al-Saady and Bennett;[34] Abraham and Yoshihara[38]).

Pulmonary Oxygen Exchange Function

In a convenience-sample study of 10 patients with severe ARDS, Abraham and Yoshihara[38] were able to show a 15% rise in mean arterial oxygen tension (P_{aO_2}) using PCV. In patients with various forms of respiratory failure, Al-Saady and Bennett[34] found a 14% increase in P_{aO_2}, and a 4% drop in the alveolar-arterial oxygen tension difference, or $P_{(A\text{-}a)O_2}$, with a decreasing-ramp pattern. Baker et al[41] were able to demonstrate only a 7% rise in P_{aO_2} but a 19% drop in $P_{(A\text{-}a)O_2}$ with the decreasing ramp, in normal dogs. In contrast, in experimentally induced emphysema, Baker et al[46] reported that the decreasing ramp produced nearly a 10% higher $P_{(A\text{-}a)O_2}$ than the constant-flow profile. In studies by Johansson and Lofstrom,[47] Johansson,[42] Lessard et al,[36] and Mang et al,[16] no statistically significant difference was observed in P_{aO_2} or shunt, between the flow patterns. However, the P_{AO_2} reported with the decreasing-ramp profile in Johansson and Lofstrom's[47] study was significantly lower than the value obtained with the rectangular pattern, so that, the calculated $P_{(A\text{-}a)O_2}$ was 24% less.

Pulmonary Carbon Dioxide Exchange Function

Only Al-Saady and Bennett,[34] Baker et al,[40,41,49] and Johansson and Lofstrom[47] reported differences in arterial carbon dioxide tension (P_{aCO_2}) or in dead-space-to-tidal volume ratio (V_D/V_T). Al-Saady and Bennett[34] reported a 16% drop in V_D/V_T when the decreasing-ramp flow was used. However, V_D/V_T was measured in only 5 of the 14 patients studied. The P_{aCO_2} was not different between the two modes for the entire sample. Baker et al[41] reported a 12% drop in V_D/V_T, and a nearly 9% decrease in P_{aCO_2}, when normal dogs were ventilated with decreasing-ramp flow. In their studies of induced pulmonary edema and pulmonary emphysema, these investigators reported that the decreasing-ramp pattern caused a 6% drop in P_{aCO_2} in the pulmonary edema group[49] and a 5% drop in both variables for the pulmonary emphysema group.[40] By contrast, Johansson and Lofstrom[47] found in anesthetized patients that P_{aCO_2} rose 9% and V_D/V_T was almost 5% higher with the decreasing-ramp pattern. When this study was repeated by Johansson[42] in patients with respiratory failure, no difference was found in measured alveolar ventilation, V_D/V_T, or P_{aCO_2}.

Critique of Gas Exchange Findings

The authors of the reviewed studies offered several reasons explaining their results and those of other investigators. Watson[2] believed that if more than 1 second was allowed for insufflation, intrapulmonary gas distribution would not be affected by inspiratory flow pattern. His work was criticized by both Baker et al[48] and Al-Saady and Bennett[34] because his ventilator was a pressure generator, so that changes in the patient's pulmonary mechanics would alter the flow patterns. However, this criticism is faulty on two points. First, Watson's ventilator used a pressurized 40-L volume reservoir.[46] According to Mushin et al,[5] a well-inflated reservoir is not subject to pressure fluctuations of clinical importance during tidal ventilation. Therefore, it can approximate a constant-flow generator. Second, in both of Watson's experiments,[1,2] study subjects were paralyzed but had normal lungs at the time of study.

A legitimate criticism leveled by Al-Saady and Bennett[34] was that Watson[2] calculated P_{aCO_2} indirectly via the Campbell rebreathing technique (probably appropriate at the time). This technique required disconnection from the ventilator for measurement and, therefore, may have been too insensitive to detect small changes in V_D/V_T.[34] More importantly, Watson's sample size (n = 3) severely limited his ability to detect any differences that may have existed.

Adams et al[45] were criticized for periodic hyperinflations used to counter the effects of progressive atelectasis (a consequence of the supine position). Al-Saady and Bennett[35] believed that this may have distorted the functional residual capacity and compliance of the animals between data runs and may have masked any potential waveform effects. Al-Saady and Bennett[34] observed that the peak inspiratory flow for the decreasing-ramp pattern studied by Adams et al[45] occurred near the middle of the inspiratory phase. This rendered it more nearly a half sine wave than a true decreasing-ramp profile (Fig. 4B). These factors may have accounted for the inability to detect differences in gas exchange.

In Johansson and Lofstrom's[47] study, a contradiction exists between reported V_T for the constant pattern (407 mL) and the decreasing-ramp pattern (411 mL) and V_T calculated from C_{dyn} and PIP (583 mL and 556 mL, respectively). This represents a 5% difference both in V_T and in minute ventilation between the two patterns (14.0 L/min vs 13.3 L/min).This could have accounted for some of the differences in P_{aCO_2}. In addition, patients in this study were ventilated with a short inspiratory time (0.6 s). Both Baker et al[41] and Watson[2] found that V_D/V_T and P_{aCO_2} rose significantly when the inspiratory time approached the range of 0.5-0.67 s. The low tidal volumes and short inspiratory times used in this study plus the high V_D/V_T (0.67-0.70) reported for patients without respiratory disease warrant cautious interpretation in terms of waveform effects.

Johansson's[42] experimental design has several deficiencies. First, with only 5 subjects, the study's ability to detect potential differences in gas exchange was hindered. Second, patients were able to assist the ventilator. The distribution of tidal ventilation changes with the change from passive to active

conditions because of differences in regional pleural pressure gradients caused by inspiratory muscle contraction.[50] This added a confounding effect on gas exchange that could not be distinguished from flow pattern effects. Third, 2/5 subjects were ventilated at frequencies that were markedly different (15 vs 24 breaths/min). Fourth, virtually no difference was reported in the peak inspiratory flows between the two patterns (66 vs 63 L/min). In addition, the decreasing-ramp pattern appeared to change at midinspiration, resembling something closer to a constant-flow pattern (Fig. 4C).

Baker et al[41] noted this change in the decreasing-ramp profile and used it to explain the differences among their results and those of Johansson and Lofstrom[47] and Johansson.[42] Baker et al[41] reasoned that if the flow patterns were substantially different, the measured $\overline{P}_{aw}$ also would have been different. It is interesting to note that the only experiments that reproduced some of the gas exchange effects reported by Baker et al[40,41,49] (Al-Saady and Bennett,[34] and Abraham and Yoshihara[38]) did not find statistically significant or clinically important differences in $\overline{P}_{aw}$ between the two waveforms. The absolute differences in $\overline{P}_{aw}$ were 0.7 cm H_2O in Al-Saady and Bennett's study and 1.0 cm H_2O in Abraham and Yoshihara's (a 5% difference). Therefore, changes in $\overline{P}_{aw}$ alone are unlikely to have accounted for differences in gas exchange in these studies. Furthermore, the waveform produced by the ventilator used by Al-Saady and Bennett incorporated a gradually decreasing-ramp waveform (Fig. 4E) rather than the more distinctive exponentially decreasing-ramp waveform of Baker et al (Fig. 4D).[48] In a later article, Baker et al[40] hypothesized that the combination of changes in $\overline{P}_{aw}$ plus the better intrapulmonary gas distribution and longer diffusion time probably accounted for the gas exchange effect seen with the decreasing-ramp pattern.

Possible differences in gas exchange effects between the two flow patterns, based on differences in $\overline{P}_{aw}$, require mention of the investigation by Mang et al.[16] This study was designed to investigate the effects of inspiratory flow pattern and the ratio of inspiratory-to-expiratory time (I:E) when the $\overline{P}_{aw}$ and total PEEP were kept constant. In addition, the PEEP level was kept above the measured P-flex point. Their results lend support to the conclusion of Boros,[18] that increase in mean pressure is responsible for the improvements in gas exchange.

Smith and Venus's[39] study of flow resistance and intrapulmonary gas distribution reported peak flows of only 9.7-17 L/min for constant and decreasing-ramp waveforms. At least in human subjects, the contribution of resistance to intrapulmonary gas distribution begins at 12 L/min[51] and becomes pronounced at 30-90 L/min.[52] Inspiratory flows used clinically, in general, range between 40 and 120 L/min.[23] Therefore, Smith and Venus[39] may have been unable to detect differences because the flows used were near the range in which intrapulmonary gas distribution becomes compliance dependent.

The problem in the study by Abraham and Yoshihara[38] was the lack of a randomized presentation of waveforms or a repeated-measures design. This may have biased their results. In Rappaport et al's study,[37] patients were able to assist the ventilator. This added the same potential confounding variable that existed in Johansson's study.[42] More importantly, the investigators remarked that the prolonged data collection period used in this study (72 hours) may have made it more difficult to detect changes in gas exchange.[37]

A design concern in the studies of Watson,[2] Rappaport et al,[37] and Lessard et al[36] was the incorporation of an end-inspiratory pause. Watson's ventilator could not eliminate the end-inspiratory pause. Rappaport et al and Lessard et al used a 10% end-inspiratory pause. An end-inspiratory pause is believed to increase intrapulmonary gas mixing and alveolar diffusion time.[53,54] Studies on the effects of a pause have shown that it can improve both oxygenation and ventilation.[53-56] However, in severe ARDS the opposite effect may occur. Lachmann et al[57] noted that during an end-inspiratory pause, the closing forces acting within the surfactant-deficient terminal lung units caused less stable alveoli to collapse into more stable ones. Any effect of waveform on gas exchange in these experiments may have been obscured by either the beneficial or detrimental effects of the end-inspiratory pause.

To summarize, 46% (6/13) of the studies that investigated the effects on gas exchange of a decreasing-ramp versus a constant inspiratory flow pattern resulted in statistically significant differences. From those 6 studies, 15 measurements of 4 variables (P_{aO_2}, $P_{(A\text{-}a)O_2}$, P_{aCO_2}, and V_D/V_T) revealed statistically significant differences. Of those measurements, 80% (12/15) indicated that decreasing-ramp flow improved gas exchange function compared to constant flow. However, most studies failed to show clinically important differences in gas exchange efficacy between the two patterns (arbitrarily set as a 10% difference). When present, distinctions in gas exchange were found in normal, obstructed, and edematous lungs, both in an animal model and in human subjects with ARDS or obstructive diseases.

Lung-Thorax Mechanics

Studies by Al-Saady and Bennett,[34] Abraham and Yoshihara,[38] and Lessard et al[36] reported statistically significant differences in PIP that ranged from 13-38% greater during constant-flow ventilation. Differences in end-inspiratory alveolar-plateau pressures were reported only by Al-Saady and Bennett, who found an 8% drop with the decreasing-ramp waveform.

Reported changes in $\overline{P}_{aw}$ between the two waveforms were conflicting. Many investigators (Rappaport et al,[37] Lessard et al,[36] Abraham and Yoshihara,[38] Al-Saady and Bennett,[34] and Adams et al[45]) reported no significant differences in $\overline{P}_{aw}$. Only Baker et al's studies[40,41,49] yielded significantly higher values for $\overline{P}_{aw}$ (20-30%) and esophageal pressures (8-20%) during decreasing-ramp ventilation. The differences in $\overline{P}_{aw}$ between decreasing-ramp and constant-flow waveforms ranged from 0.3 to 1.2 cm H_2O. In the clinical studies, these differences

constituted only a 5% change. Only in Baker's studies,[40,41,49] in which total $\bar{P}_{aw}$ was less (3.87 and 5.03 cm H_2O), did a difference of 1.2 cm H_2O assume greater importance.

Al-Saady and Bennett[34] and Abraham and Yoshihara[38] reported the most salient changes in compliance. Al-Saady and Bennett found significantly higher static (17%) and kinetic (15%) lung-thorax compliances with a decreasing-ramp waveform. The calculation of kinetic compliance required the measurement of the pressure drop associated with tissue resistance during inflation. Al-Saady and Bennett reported significantly lower pressures associated with tissue resistance and maldistribution of ventilation when the decreasing-ramp pattern was used. In addition, total resistance was 31% lower with the ramp profile. Abraham and Yoshihara found a 35% improvement in C_{dyn} in ARDS patients. Rappaport et al[37] reported that inspiratory resistance was 30% lower during PCV in nonsurvivors but indistinguishable in survivors. C_{dyn} measured by Baker et al[41] and Johansson[42] was significantly higher with a decreasing-ramp waveform.

Critique of Lung-Thorax Mechanics Findings

The theoretical constructs predict a primary improvement in $C_{LT\text{-}stat}$ in ARDS and an improvement in C_{dyn} in both ARDS and obstructive diseases.[21,25] Static measurements were made only by Al-Saady and Bennett[34] and Rappaport et al.[37] Al-Saady and Bennett's findings of an improvement in $C_{LT\text{-}stat}$ and C_k with the decreasing-ramp pattern support the theoretical propositions. Rappaport et al reported that $C_{LT\text{-}stat}$ "improved more rapidly" with PCV in ARDS, but these results failed to reach statistical significance ($p = 0.05$). Although most studies that measured C_{dyn} found that it improved with the decreasing-ramp pattern, it is not possible to discern how much of that improvement was caused by the reapportionment of gas flow resistance and how much, if any, could be attributed to lessened tissue resistance and better distribution. Al-Saady and Bennett's measurements of C_k and total resistance appear to support the idea that the decreasing-ramp profile improves the distribution of tidal ventilation and decreases tissue resistance.

Cardiovascular-Cardiorespiratory Variables

In the nine studies reporting cardiovascular and cardiorespiratory variables, no significant differences were found. The increase in systemic oxygen delivery reported by Abraham and Yoshihara[38] was attributed to the increase in oxygen saturation alone. Because no statistically significant differences were found in any study, regardless of methods or subjects, a critique of the methodology was not undertaken.

Interpreting the Experimental Findings

The experimental results reported in this overview are problematic. First, attempting to compare changes in gas exchange and lung mechanics, on the basis of predicted effects, produced mixed results. The only consistent findings between predicted changes in gas exchange and lung mechanics were those reported by Al-Saady and Bennett[34] and Baker et al.[41] In both studies, the improvement in CO_2-exchange function coincided with an increased C_{dyn}. Although the increase in oxygenation found by Al-Saady and Bennett was associated with an improvement in $C_{LT\text{-}stat}$, there is no way to know whether the improvements in P_{aO_2} and P_{aCO_2} were caused by lung recruitment or better distribution of ventilation or both. The improvement in CO_2 elimination reported by Baker et al[40,49] and by Johansson and Lofstrom[47] did not coincide with improvements in C_{dyn}. The ARDS study by Abraham and Yoshihara,[36] in which an impressive improvement in P_{aO_2} was seen, reported only total C_{dyn}. Therefore, it is not possible to discern whether lung recruitment lessened tissue resistance or better distribution occurred.

Second, the decreasing-ramp flows employed in most of these studies differed from one another. This may partially explain why the effects of a decreasing-ramp flow pattern on gas exchange were inconsistent. The decreasing-ramp flow pattern produced by the ventilator in Adams et al's[45] study was closer to a half sine wave. In Johannson's studies,[42,47] the waveform appeared more like a hybrid pattern in which the decreasing ramp stabilized its flow after mid-inspiration. What may be more important is that the glaring disparity between the published flow contours and the reported differences in peak flows (5-15%) undermines the credibility of the studies. Abraham and Yoshihara,[38] Lessard et al,[36] and Mang et al[16] did not publish flow tracings. Given the ventilator all groups used (Siemens Servo 900C) and the low lung compliance of the study subjects, the flow pattern during PCV likely was an exponentially decreasing-ramp profile. Smith and Venus[39] appeared to have used an exponentially decreasing waveform available in VCV on the Veolar ventilator. It should be noted that significant gas exchange effects were produced, both with rapid and with gradually decreasing-ramp patterns.

Third, questions can be raised concerning the performance capabilities of modern commercial ventilators versus older experimental models. Watson,[46] Adams et al,[45] and Wilson and Baker [58] used experimental ventilators. Watson's ventilator appears to have been a time-cycled, pressure generator. The design was conceptually similar to the Siemens Servo, in that a pressurized volume reservoir was connected to an inspiratory valve with an adjustable orifice. Its waveforms were created by a rotating ("tear drop") disk. The disk's movement during each profile was controlled by custom-designed cams. Therefore, tidal volume and inspiratory time were not affected by changes in flow or pressure pattern. In terms of performance, during rectangular pressure-profile ventilation, peak inspiratory flows in excess of 100 L/min could be delivered, and the PIP could be reached in 0.1 seconds.[46]

Wilson and Baker's ventilator[58] was an electronically controlled, time-cycled, flow generator that had a driving pres-

sure of 85 psi and could reach peak inspiratory flows of 160 L/min. In terms of performance, the ventilators used by Watson et al and Baker et al do not appear to be mechanically inferior (relative to the needs of the studies). Only Adams et al[45] did not provide information about their ventilator. Given the poor quality of the decreasing-ramp waveform it produced, one may wish to disregard this study on that basis alone.

Fourth, a question can be raised about the relationship between PEEP and the P-flex point in patients with ARDS. The decreasing-ramp flow pattern may be more effective than the rectangular-flow pattern for improving oxygenation when PEEP is below this point. The higher initial pressure step, more rapid rise in alveolar pressure, and volume equilibration may provide for faster recruitment of collapsed alveoli. This may be the critical difference in oxygenation between the two waveforms when PEEP is below the P-flex point. However, if PEEP is at or above the P-flex point, then the flow pattern may not be significant.

Fifth, because collateral ventilation is much greater in dogs than in human subjects, a concern arises related to the results of dog studies.[59] This animal model has been criticized for having questionable applicability to humans.[60] It is possible that the positive effects of the decreasing-ramp waveform reported by Baker et al,[40,41,49] may have been potentiated by enhanced collateral ventilation present in the dogs.

Sixth, the studies in this review had too few subjects, considering the investigators' decisions to use parametric statistical tests. The validity of parametric tests rests upon the assumption that a normal distribution of measurement values exists within the sample. This assumption (the Central Limit Theorem) is increasingly susceptible to violation when the sample size falls below 30.[61] In addition, the ability or power of a statistical test to discern a treatment effect between groups depends upon the sample size.[62] When the sample size is small (< 30) the ability to detect a treatment effect (that, in fact, exists) becomes harder. Unless the treatment effect is very pronounced, experiments carried out with samples of small size generally will not detect statistically significant differences,[63] and, therefore, experimental results taken from such studies that show no difference between treatment groups are not conclusive. If more subjects had been studied a treatment effect might have been found.

A general limitation in clinical research is the ethical constraint to study patients only when they are relatively stable. At my institution, where PCV is used in the majority of severe respiratory failure cases, we have anecdotal reports of a number of extremely ill patients who have shown improvement and stabilization of gas exchange after being switched from VCV to PCV. I entered some of these patients into a study of the relative gas exchange effects of VCV and PCV, once they were clinically stable. However, I was never able to reproduce the beneficial effects of PCV observed when these same patients were unstable [unpublished study]. The two possible explanations for this are (1) in an uncontrolled setting, when patients are unstable, a number of dynamic and occult confounding variables (such as changes in pulmonary perfusion or the effects of sedation, inotropes, or diuretics) may have accounted for some of the changes that were attributed to PCV alone; (2) PCV may, in fact, have more clinical efficacy in unstable states precisely when controlled study is impossible.

Owen-Thomas et al[64] studied the effects of inspiratory flow on arterial oxygenation in neonates suffering from idiopathic respiratory distress syndrome (IRDS). They found that an optimal inspiratory flow (in terms of oxygenation) was both "specific and reproducible for a given patient at a given time." However, this optimal flow varied temporally during the disease course and could only be arrived at empirically, not by rational prediction. Because of pathophysiologic similarities between IRDS and ARDS in the mechanical disturbances,[65] the observations by Owen-Thomas et al[64] may help to explain the effects of PCV observed in unstable patients with ARDS. The study of Abraham and Yoshihara[38] came closest to describing this situation.

Temporal changes in lung mechanics, relative to time of study, may play a role in determining the effects of different waveforms on gas exchange. Klose and Osswald[66] observed that PEEP produced no obvious recruitment of lung tissue and only minimal improvement in P_{aO_2} in the advanced stages (fibrotic or consolidated) of ARDS. They found that P_{aO_2} increased only 2 torr/cm H_2O PEEP. This represents a 1-cm-H_2O change in $\bar{P}_{aw}$, which is the same difference generally reported between the constant-flow and decreasing-ramp waveforms. If change in $\bar{P}_{aw}$ is the primary factor affecting gas exchange, then no significant differences would be expected in these patients. Therefore, the mix of ARDS patients in these studies, between early and late stage, could have influenced study outcomes. As an example, in Lessard et al's study,[36] 44% (4/9) of the sample had been ventilated 10-16 days.

In Conclusion

No definitive statement can be made regarding the superiority of either flow pattern over the other. The only consistent result across all studies was that cardiovascular performance was not affected differently by either flow pattern. The inability to draw strong inferences from the clinical and/or laboratory evidence stems from the following limitations and potential confounding influences: (1) the choice of dogs as the experimental model (Adams et al,[45] Baker et al[40,41,49]); (2) the choice of ventilator settings, such as low V_T and short inspiratory time (Johansson and Lofstrom[47]), inconsistent rate and inspiratory time across the sample (Johansson[42]), use of a post-inspiratory pause (Watson,[2] Rappaport et al,[37] Lessard et al[36]), insignificant changes in peak flows (Johansson and Lofstrom,[47] Johansson[42]), and clinically irrelevant flows (Smith and Venus[39]); (3) the presence of morphologic differences in the decreasing-ramp pattern (Adams et al[45]); (4) the design employed, such as absence of randomized presentation or repeated-

measures (Abraham and Yoshihara[38]); (5) the small number of subjects (all studies); and (6) failure to segregate sample by disease process (Johansson,[42] Al-Saady and Bennett[34]). Therefore, the definitive clinical study (or studies) on the relative effects of constant versus decreasing-ramp flow has yet to be undertaken. The number of patients in any future study should approach 30 to allow application of a parametric test or 60 for true randomization. This study should segregate its sample based on primary mechanical disturbances (resistive vs elastic). In the case of ARDS, the sample should be segregated further according to early versus late stage. In addition, better qualitative reporting on the type of pulmonary lesions present in each patient (ie, diffuse, unilateral, lobar, or 'patchy' infiltrates; consolidation, atelectasis or pleural effusions) might help to clarify those situations in which different flow patterns may have efficacy. The study should be performed without an inspiratory pause, and peak inspiratory flows should be reported for each waveform. Also, it would be informative to look at the relative effects of these waveforms when PEEP is adjusted below and at the P-flex point.

With the inconsistent results presented, how is the practitioner to apply these waveforms in clinical practice? The fact that treatment effects, when found, were relatively modest suggests that inspiratory flow waveforms do not effect dramatic changes in gas exchange (relative to manipulations in F_{IO_2}, minute ventilation, or PEEP). Despite the methodologic problems (or concerns) that exist in most of the studies reviewed, it is still reasonable to anticipate that individual patients with pulmonary disease may show modest-to-moderate improvements in pulmonary gas exchange when the decreasing-ramp pattern is used. The majority of studies that demonstrate statistically significant results support this interpretation.

If I base my position on theory, the patients who might benefit from a decreasing-ramp waveform are those with early stages of diffuse ARDS (in which the primary derangements are interstitial edema and congestive atelectasis) and those with obstructive lung disease. In unilateral lung disease or when lung disease is marked by patchy infiltrates, a constant-flow pattern may be superior. However, the clinician's decision to use any flow pattern in the treatment of respiratory disease should be made empirically based on specific evidence, not a priori.

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Control of Pneumocystis Infections

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Background

Before 1980, *Pneumocystis carinii* was an uncommon human pathogen, an opportunistic organism that was the terminal invader in patients at the extremes of age—the neonate with undeveloped lungs and the aged person racked with debilitating illness. In the United States, fewer than 100 cases were reported annually.[1] Interest in the organism itself was casual; even its identity—fungus or protozoan?—was in doubt for several decades after its discovery.[2]

The clinical importance of *P. carinii* emerged with the onset of the acquired immunodeficiency syndrome (AIDS) epidemic in the early 1980s. Today it is the most common cause of mortality in AIDS victims.[3] Added to this susceptible group are the growing numbers of patients immunosuppressed by anticancer drugs and agents used to prevent rejection of organ transplants. The annual incidence of *P. carinii* infections reported in the U.S.A. now stands at about 50,000 cases and appears to be increasing despite the use of prophylactic medications.[3]

P. carinii is now classified as a fungus,[2] although some features of its life cycle are still unknown. It is a more common invader in AIDS patients than are other fungi, such as candida, cryptococcus, and aspergillus. It most often affects the lungs. Disseminated infections are unusual and represent a nearly total loss of host resistance. The organism binds to type-1 pneumocytes and damages the alveolocapillary membrane, increasing its permeability and causing the alveolus to fill with foamy exudate. The resulting inflammatory reaction is accompanied by proliferation of type-2 pneumocytes and fibroblasts.[4] Gas exchange is progressively impaired as surfactant loss, exudate accumulation,[5] and eventual parenchymal destruction result in widespread pulmonary fibrosis. The clinical course is commonly indolent, especially if a prophylactic regimen is in use, but the course may be fulminating in seriously immunodepressed, untreated patients, in whom the mortality approaches 100%.[6]

Current Therapy & Prophylaxis

Pentamidine

In the years prior to the taxonomic identification of *P. carinii*, antiprotozoan agents were employed to treat pneumocystis infections. Among these were pentamidine and stilbamidine; pentamidine was found to be the more effective and is still in use despite its marked systemic toxicity. Pentamidine has been given by aerosol in an effort to minimize its effects on organs other than the lungs; however, this protection is incomplete and serious side effects can occur.[7-9] In the period prior to 1980, pentamidine was the drug of choice for *P. carinii* pneumonia.[10]

Folate Enzyme Inhibitors

Subsequent investigations showed that *P. carinii*, like many microorganisms, cannot utilize preformed folic acid derivatives taken from the environment. These compounds, essential to cell replication, must be synthesized from metabolic precursors.[11] This is in contrast to mammalian cells, which lack the folate biosynthetic enzymes and must take folate from dietary sources. One important enzyme in microbial synthesis of folate is dihydropteroate synthase, which is inhibited by some sulfonamides. However, most of these well-known compounds have been found to be only weakly effective against *P. carinii*.

A second enzyme of importance in folate synthesis, dihydrofolate reductase, has been isolated in crystalline form. This enzyme is present in both mammals and microorganisms, but there are enough structural differences between the mammalian and microbial forms to allow drug inhibition in microbes selectively.[12] Certain antimalarial drugs can accomplish this selective inhibition, notably pyrimethamine and trimethoprim. When combined with sulfonamides the suppression of the two enzymes is sufficient to deprive *P. carinii* organisms of folate and to arrest the progress of lung infections. The most widely accepted combination is trimethoprim and sulfamethoxazole (eg, generics, Bactrim, Septra);[13] another is pyrimethamine and sulfadoxine (Fansidar).[14] These combinations were exten-

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sively prescribed for other infections before their efficacy against the *Pneumocystis* species became known.[15]

A considerably more potent inhibitor of dihydrofolate reductase is the anticancer drug methotrexate. However, it inhibits not only the microbial enzyme but also the human one, the property that accounts for its carcinocidal action. Methotrexate, thus, cannot be utilized for *P. carinii* infections without serious risk of bone marrow suppression and viscerotoxicity.

The foregoing compounds with established efficacy have provided researchers with leads toward the synthesis of new drugs that offer greater therapeutic promise and reduced toxicity. An overview of these research avenues has recently been published in a comprehensive article by Queener.[16] The author points out that all animal models for *P. carinii* pneumonia require weeks and large quantities of drugs to carry out suitable investigations. This accounts in part for slow progress; some newer technical developments are described that may accelerate screening of compounds and broaden the scope of purview of chemical structures.[16]

New Antifolates

The first enzyme described earlier, dihydropteroate synthase, has recently been isolated, and the gene encoding the enzyme has been cloned.[17] No promising new sulfonamides have yet appeared, but a related sulfone compound, dapsone, has undergone clinical trials. This is an old drug, first advanced for use against tuberculosis and later for leprosy and certain skin conditions. A trimethoprim-dapsone combination has been shown to be as effective as trimethoprim-sulfamethoxazole, with purportedly less-serious toxic side effects.[18] At least one new sulfone is now (1996) under study.[19]

Numerous dihydrofolate reductase inhibitors have been prepared. Those related to trimethoprim (tetroxoprim, epiroprim[20]) and to pyrimethamine (cycloguanil[16]) are of limited potency and are effective only when combined with sulfonamides. Those related to methotrexate (trimetrexate,[21] piritrexim,[22] edetrexate[16]) are potent and effective alone but are, like the parent compound, limited by toxicity. Derivatives of each of these have been screened, but the ultimate goal, freedom from inhibition of the human enzyme, has yet to be achieved. Recent crystallographic studies of the enzyme molecule, both isolated and in combination with various inhibitors, have greatly enlarged the scope of new compounds with novel ring systems and substituent chemical groups.[16]

Agents Affecting DNA Binding or Conformation

This rather large category of antimicrobial and anticancer compounds is currently the major field of exploration to develop agents against *P. carinii*. Pentamidine and related compounds probably belong in this category, although the specific mode of action is unknown.[23] A number of these are under investigation,[24] but a common drawback to their eventual clinical use is that they must be given parenterally. Fluoroquinolones (ciprofloxacin, norfloxacin, others) also inhibit DNA conformation, but studies have failed to yield promising antipneumocystis compounds.[25]

Antimalarials

Although *P. carinii* is not a protozoan, a number of compounds originally introduced as antimalarials have demonstrable efficacy. Pyrimethamine and trimethoprim have already been noted. Primaquine, combined with clindamycin, is effective against *P. carinii* in animal models.[26] Atovaquone, a hydroxynaphthoquinone[27] with a broad antiprotozoan spectrum, is currently in clinical use for treatment of pneumocystis infection.[28] Although less effective than the combination of trimethoprim and sulfamethoxazole, its shortcomings appear to be pharmacokinetic, and a prodrug has been developed to improve bioavailability and make absorption more uniform.[29] It is postulated that an intracellular oxidative process common to eukaryocytes may be responsible for the antifungal and antiprotozoan action of these agents.

Summary

The urgency for treatment of AIDS patients has stimulated the investigation of established and novel drugs in both antifungal and antiprotozoan categories for possible use in prevention and treatment of *P. carinii* pneumonia. Although few of the older drugs have proven more than marginally effective, these trials have promoted great interest in related families of chemical congeners. The search for antipneumocystis agents may eventually be of benefit in treatment of tropical protozoan diseases and for opportunistic fungal infections common to immunosuppressed patients.

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42nd International Convention & Exhibition

SAN DIEGO

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The American Association for Respiratory Care Clinical Practice Guidelines

- **Spirometry, 1996 Update**
- **Selection of an Oxygen Delivery Device for Neonatal and Pediatric Patients**
- **Selection of a Device for Delivery of Aerosol to the Lung Parenchyma**
- **Training the Health-Care Professional for the Role of Patient and Caregiver Educator**
- **Providing Patient and Caregiver Training**

Respir Care 1996;41(7):629-663

Previously Published Guidelines:

- Assessing Response to Bronchodilator Therapy at Point of Care
- Discharge Planning for the Respiratory Care Patient
- Long-Term Invasive Mechanical Ventilation in the Home
- Capnography/Capnometry during Mechanical Ventilation
- Selection of an Aerosol Delivery Device for Neonatal and Pediatric Patients
- Polysomnography

Respir Care 1995;40(12):1300-1343

- Defibrillation during Resuscitation
- Management of Airway Emergencies
- Infant/Toddler Pulmonary Function Tests

Respir Care 1995;40(7):744-768

- Metabolic Measurement Using Indirect Calorimetry during Mechanical Ventilation
- Transcutaneous Blood Gas Monitoring for Neonatal and Pediatric Patients
- Capillary Blood Gas Sampling for Neonatal and Pediatric Patients
- Body Plethysmography

Respir Care 1994;39(12):1170-1190

- Ventilator Circuit Changes
- Delivery of Aerosols to the Upper Airway
- Neonatal Time-Triggered, Pressure-Limited, Time Cycled Mechanical Ventilation
- Application of Continuous Positive Airway Pressure to Neonates via Nasal Prongs or Nasopharyngeal Tube
- Surfactant Replacement Therapy
- Static Lung Volumes

Respir Care 1994;39(8):797-836

- Transport of the Mechanically Ventilated Patient
- Fiberoptic Bronchoscopy Assisting
- Resuscitation in Acute Care Hospitals
- Intermittent Positive Pressure Breathing
- Bland Aerosol Administration

Respir Care 1993;38(11):1169-1200

- Directed Cough
- Endotracheal Suctioning of Mechanically Ventilated Adults and Children with Artificial Airways
- In-Vitro pH and Blood Gas Analysis and Hemoximetry
- Single-Breath Carbon Monoxide Diffusing Capacity
- Use of Positive Airway Pressure Adjuncts to Bronchial Hygiene Therapy

Respir Care 1993;38(5):495-521

- Patient-Ventilator System Checks
- Humidification during Mechanical Ventilation
- Selection of Aerosol Delivery Device
- Nasotracheal Suctioning
- Bronchial Provocation
- Exercise Testing for Evaluation of Hypoxemia and/or Desaturation
- Arterial Blood Gas Sampling
- Oxygen Therapy in the Home or Extended Care Facility

Respir Care 1992;37(8):855,856,882-922

- Incentive Spirometry
- Pulse Oximetry
- Oxygen Therapy in the Acute Care Hospital
- Spirometry
- Postural Drainage Therapy

Respir Care 1991;36(12):1398-1426

AARC Clinical Practice Guideline

Spirometry, 1996 Update

S 1.0 PROCEDURE:

Spirometry (S): The first American Association for Respiratory Care (AARC) Spirometry Clinical Practice Guideline,[1] published in 1991, was based largely on the American Thoracic Society (ATS) 1987 recommendations.[2] Since that time, the ATS has published new recommendations.[3] This updated AARC Clinical Practice Guideline not only reflects these new ATS recommendations but also contains additional recommendations on the use of bronchodilators in conjunction with spirometry.

S 2.0 DESCRIPTION/DEFINITION:

The objective of spirometry is to assess ventilatory function. Spirometry includes but is not limited to the measurement of forced vital capacity (FVC), the forced expiratory volume in the first second (FEV_1), and other forced expiratory flow measurements such as the $FEF_{25-75\%}$. In addition, it sometimes includes the measurement of maximum voluntary ventilation (MVV). A graphic representation (spirogram) of the maneuver should be a part of the results. Either a volume-time or flow-volume display is acceptable. Other parameters that may be obtained by spirometry include FEF_{max} (PEF), $FEF_{75\%}$, $FEF_{50\%}$, $FEF_{25\%}$, $FIF_{50\%}$, and FIF_{max} (PIF).

S 3.0 SETTING:

These guidelines should be applied to spirometry performed by trained health-care professionals

3.1 in the pulmonary function or research laboratory;

3.2 at the bedside, in acute, subacute, extended care, and skilled nursing facilities;

3.3 in the clinic, treatment facility, and physician's office;

3.4 in the workplace or home;

3.5 for public screening.

S 4.0 INDICATIONS:

The indications for spirometry[4-8] include the need to

4.1 detect the presence or absence of lung dysfunction suggested by history or physical signs and symptoms (eg, age, smoking history, family history of lung disease, cough, dyspnea, wheezing) and/or the presence of other abnormal diagnostic tests (eg, chest radiograph, arterial blood gas analysis);

4.2 quantify the severity of known lung disease;

4.3 assess the change in lung function over time or following administration of or change in therapy;

4.4 assess the potential effects or response to environmental or occupational exposure;

4.5 assess the risk for surgical procedures known to affect lung function;

4.6 assess impairment and/or disability (eg, for rehabilitation, legal reasons, military).

S 5.0 CONTRAINDICATIONS:

The requesting physician should be made aware that the circumstances listed in this section could affect the reliability of spirometry measurements. In addition, forced expiratory maneuvers may aggravate these conditions, which may make test postponement necessary until the medical condition(s) resolve(s).

Relative contraindications[9,10] to performing spirometry are

5.1 hemoptysis of unknown origin (forced expiratory maneuver may aggravate the underlying condition);

5.2 pneumothorax;

5.3 unstable cardiovascular status (forced expiratory maneuver may worsen angina or cause changes in blood pressure) or recent myocardial infarction or pulmonary embolus;

5.4 thoracic, abdominal, or cerebral aneurysms (danger of rupture due to increased thoracic pressure);
5.5 recent eye surgery (eg, cataract);
5.6 presence of an acute disease process that might interfere with test performance (eg, nausea, vomiting);
5.7 recent surgery of thorax or abdomen.

S 6.0 HAZARD/COMPLICATIONS:

Although spirometry is a safe procedure, untoward reactions may occur, and the value of the information anticipated from spirometry should be weighed against potential hazards. The following have been reported anecdotally:

6.1 pneumothorax;
6.2 increased intracranial pressure;
6.3 syncope, dizziness, light-headedness;
6.4 chest pain;
6.5 paroxysmal coughing;
6.6 contraction of nosocomial infections;
6.7 oxygen desaturation due to interruption of oxygen therapy;
6.8 bronchospasm.

S 7.0 LIMITATIONS OF METHODOLOGY/ VALIDATION OF RESULTS:

7.1 Spirometry is an effort-dependent test that requires careful instruction and the cooperation of the test subject. Inability to perform acceptable maneuvers may be due to poor subject motivation or failure to understand instructions. Physical impairment and young age (eg, children < 5 years of age) may also limit the subject's ability to perform spirometric maneuvers. These limitations do not preclude attempting spirometry but should be noted and taken into consideration when the results are interpreted.

7.2 The results of spirometry should meet the following criteria for number of trials, acceptability, and reproducibility. The acceptability criteria should be applied before reproducibility is checked.

7.2.1 Number of trials: A minimum of 3 acceptable FVC maneuvers should be performed.[3] If a subject is unable to perform a single acceptable maneuver after 8 attempts, testing may be discontinued. However, after additional instruction and demonstration, more maneuvers may be performed depending on the subject's clinical condition and tolerance.

7.2.2 Acceptability: A good 'start-of-test' includes:

7.2.2.1 an extrapolated volume of ≤ 5% of the FVC or 150 mL, whichever is greater;
7.2.2.2 no hesitation or false start;
7.2.2.3 a rapid start to rise time.

7.2.3 Acceptability: no cough, especially during the first second of the maneuver.

7.2.4 Acceptability: no early termination of exhalation.

7.2.4.1 A minimum exhalation time of 6 seconds is recommended, unless there is an obvious plateau of reasonable duration (ie, no volume change for at least 1 second) or the subject cannot or should not continue to exhale further.[3]

7.2.4.2 No maneuver should be eliminated solely because of early termination. The FEV_1 from such maneuvers may be valid, and the volume expired may be an estimate of the true FVC, although the FEV_1/FVC and $FEF_{25-75\%}$ may be overestimated.

7.2.5 Reproducibility:

7.2.5.1 The two largest FVCs from acceptable maneuvers should not vary by more than 0.200 L, and the two largest FEV_1s from acceptable maneuvers should not vary by more than 0.200 L.

Note: The ATS has changed its recommendations from those made in the 1987 ATS guideline[2] (a reproducibility criterion of 5% or 0.100 L, whichever is larger). This change is based on evidence from Hankinson and Bang suggesting that intrasubject variability is independent of body size and that individuals of short stature are less likely to meet the older criterion than are taller subjects.[11] In addition, the 0.200-L criterion is simple to apply. However, there are two concerns with this change. The first is whether the 0.200-L criterion is too permissive in shorter individuals (eg, children). Enright and co-workers[12] reported a failure rate of only 2.1% in 21,432 testing

sessions on adults using the 5% or 100-mL criterion. In addition, they found that only 0.4% of test sessions failed to meet relaxed criteria of 5% or 200 mL. These failure rates are much lower than the 5-15% failure rates reported by Hankinson and Bang.[11] Enright and co-workers did not study children, but there was some height overlap in the two studies. Thus, we are not convinced that the 5% rule is inappropriate when applied to shorter individuals. Indeed, Hankinson and Bang stated in their report "...it appears that the technician appropriately responded to the lack of a reproducible or acceptable test result by obtaining more maneuvers from these subjects." The second concern is that the 0.200-L criterion may be too rigid for very tall individuals (eg, height > 75 inches). Hankinson and Bang did not study subjects taller than 190 cm (ie, 75 inches). In order to send a consistent message, we recommend the ATS reproducibility criterion but urge practitioners: (a) to use this criterion as a goal during data collection and not to reject a spirogram solely on the basis of its poor reproducibility, (b) to exceed the reproducibility criterion whenever possible because it will decrease inter- and intralaboratory variability, and (c) to comment in the written report when reproducibility criteria cannot be met.

7.3 Maximum voluntary ventilation (MVV) is the volume of air exhaled in a specified period during rapid, forced breathing.[3] This measurement is sometimes referred to as the maximum breathing capacity (MBC).

7.3.1 The period of time for performing this maneuver should be at least 12 seconds but no more than 15 seconds, with the data reported as L/min at BTPS.

7.3.2 At least two trials should be obtained, and the two highest should agree within ±10%.

7.4 The use of a nose clip for all spirometric maneuvers is strongly encouraged.

7.5 Subjects may be studied in either the sitting or standing position. Occasionally, a subject may experience syncope or dizziness while performing the forced expiratory maneuver. Thus, the sitting position may be safer. If such a subject is standing, an appropriate chair (ie, with arms and not on rollers) should be placed behind the subject in the event that he or she needs to be seated quickly. When the maneuver is performed from a seated position, the subject should sit erect with both feet on the floor, and be positioned correctly in relation to the equipment. Test position should be noted on the report.

7.6 Spirometry is often performed before and after inhalation of a bronchodilator.

7.6.1 The drug, dose, and mode of delivery should be specifically ordered by the managing physician or determined by the laboratory and should be noted in the report.

7.6.2 The length of the interval between administration of the bronchodilator and postbronchodilator testing varies among laboratories,[13-17] but there appears to be more support for a minimum interval of 15 minutes for most short and intermediate-acting β-2 agonists.[14-17] This does not guarantee that peak response will be determined, and underestimation of peak bronchodilator response can occur.

7.6.3 Subjects who use inhaled short-acting bronchodilators should be tested at least 4 to 6 hours after the last use of their inhaled bronchodilator to allow proper assessment of acute bronchodilator response. Long-acting inhaled bronchodilators may need to be withheld for a more extended period. Subjects should understand that if they need to administer their bronchodilator prior to the test because of breathing problems, they should do so. Bronchodilators taken on the day of testing should be noted in the report. Table 1 lists commonly used drugs that may confound assessment of acute bronchodilator response and the recommended times for withholding.

7.6.4 Interpretation of response to a bronchodilator should take into account both magnitude and consistency of change in the pulmonary function data. The recommended criterion for response to a bronchodilator in adults for FEV_1 and FVC is a 12% improvement from baseline *and* an absolute change of 0.200 L.[18] However, because the peak effect of the drug may

Table 1. Recommended Times for Withholding Commonly Used Bronchodilators When Bronchodilator Response Is To Be Assessed*

Drug	Withholding Time (hours)
salmeterol	12
ipratropium	6
terbutaline	4-8
albuterol	4-6
metaproterenol	4
isoetharine	3

*Based on consensus of committee and known duration of action

not always be determined, the inability to meet this response criterion does not exclude a response. In addition, dynamic compression of the airways during the forced expiratory maneuver may mask bronchodilator response in some subjects, and the additional measurement of airway resistance and calculation of specific conductance and resistance may provide documentation of airway responsiveness.[19]

7.7 Reporting of results:

7.7.1 The largest FVC and FEV_1 (at BTPS) should be reported even if they do not come from the same curve.

7.7.2 Other reported measures (eg, $FEF_{25-75\%}$ and instantaneous expiratory flowrates, such as FEF_{max} and $FEF_{50\%}$) should be obtained from the single acceptable 'best-test' curve (ie, largest sum of FVC and FEV_1) and reported at BTPS.

7.7.3 All values should be recorded and stored so that comparison for reproducibility and the ability to detect spirometry-induced bronchospasm (as evidenced by a worsening in spirometric values with successive attempts—and not related to fatigue) are simplified.

7.7.4 The highest MVV trial should be reported.

7.8 Subject demographics and related information:

7.8.1 Age: The age on day of test should be used.

7.8.2 Height: The subject should stand fully erect with eyes looking straight ahead and be measured with the feet together without shoes. An accurate measuring device should be used. For subjects who cannot stand or who have a spinal deformity (eg, kyphoscoliosis), the arm span from finger tip to finger tip with arms stretched in opposite directions can be used as an estimate of height.[20]

7.8.3 Weight: An accurate scale should be used to determine the subject's weight while wearing indoor clothes but without shoes.

7.8.4 Race: The race or ethnic background of the subject should be determined and reported to help ensure the use of appropriate reference values and appropriate interpretation of data.

7.8.5 The time of day, equipment or instrumentation used, and name of the technician administering the test should be recorded.

7.9 Open- and closed-circuit testing:

7.9.1 Open circuit: The subject takes a maximal inspiration from the room, inserts the mouthpiece into the mouth, and then blows out either slowly (SVC) or rapidly (FVC) until the end-of-test criterion is met. Although the open-circuit technique works well for some subjects, others have difficulty maintaining a maximum inspiration while trying to position the mouthpiece correctly in the mouth. These subjects may lose some of their vital capacity due to leakage prior to the expiratory maneuver.[11]

7.9.2 Closed-circuit: The subject inserts the mouthpiece into the mouth and breathes quietly for no more than 5 tidal breaths, takes a maximal inspiration from the reservoir, and then blows out either slowly (SVC) or rapidly (FVC) until the end-of-test criterion is met. This rebreathing technique is preferred if the spirometer system permits because it (1) allows the subject to obtain a tight seal with the mouthpiece prior to inspiration and (2) allows evaluation of the volume inspired.

S 8.0 ASSESSMENT OF NEED:

Need is assessed by determining that valid indications are present.

S 9.0 ASSESSMENT OF TEST QUALITY:

Spirometry performed for the listed indications is valid only if the spirometer functions acceptably and the subject is able to perform the maneuvers in an acceptable and reproducible fashion. All reports should contain a statement about the technician's assessment of test quality and specify which acceptability criteria were not met.

9.1 Quality control:[21]

9.1.1 Volume verification (ie, calibration): at least daily prior to testing, use a calibrated known-volume syringe with a volume of at least 3 L to ascertain that the spirometer reads a known volume accurately. The known volume should be injected and/or withdrawn at least 3 times, at flows that vary between 2 and 12 L/s (3-L injection times of approximately 1 second, 6 seconds, and somewhere between 1 and 6 seconds). The tolerance limits for an acceptable calibration are ± 3% of the known volume. Thus, for a 3-L calibration syringe, the acceptable recovered range is 2.91-3.09 L. We encourage the practitioner to exceed this guideline whenever possible (ie, reduce the tolerance limits to < ± 3%)

9.1.2 Leak test: Volume-displacement spirometers must be evaluated for leaks daily. One recommendation is that any volume change of more than 10 mL/min while the spirometer is under at least 3-cm-H_2O pressure be considered excessive.[22]

9.1.3 A spirometry procedure manual should be maintained.

9.1.4 A log that documents daily instrument calibration, problems encountered, corrective action required, and system hardware and/or software changes should be maintained.

9.1.5 Computer software for measurement and computer calculations should be checked against manual calculations if possible. In addition, biologic laboratory standards (ie, healthy, nonsmoking individuals) can be tested periodically to ensure historic reproducibility, to verify software upgrades, and to evaluate new or replacement spirometers.

9.1.6 The known-volume syringe should be checked for accuracy at least quarterly using a second known-volume syringe, with the spirometer in the patient-test mode. This validates the calibration and ensures that the patient-test mode operates properly.

9.1.7 For water-seal spirometers, water level and paper tracing speed should be checked daily. The entire range of volume displacement should be checked quarterly.[21]

9.2 Quality Assurance: Each laboratory or testing site should develop, establish, and implement quality assurance indicators for equipment calibration and maintenance and patient preparation. In addition, methods should be devised and implemented to monitor technician performance (with appropriate feedback) while obtaining, recognizing, and documenting acceptability criteria.

S 10.0 RESOURCES:

10.1 Equipment: The spirometer must meet or exceed the requirements proposed by the ATS and must be calibrated appropriately.[3] Spirometers should produce a paper record of volume-time and/or flow-volume displays. Reference values should be appropriate for the population of subjects tested and should be validated by testing a group of healthy, nonsmoking subjects with the same mix of age, gender, and height used in the reference study.[18]

10.2 Personnel: Spirometry should be administered under the direction of a doctor (MD, DO, or PhD) specifically trained in pulmonary function testing. The value of spirometry results can be compromised by poor patient instruction secondary to inadequate technician training. Thus, technicians should have documented training, with continued competency assessments in spirometry administration and recognition of causes for errors encountered in the testing process and a sound understanding of physiologic effects caused by bronchodilators. They should be trained in basic life support and emergency procedures appropriate to the setting.[23] Spirometry can be administered by persons who meet criteria for either Level I or Level II.

10.2.1 Level I: Persons trained in and with demonstrated ability to perform spirometry. Minimum educational requirements for Level-I personnel should be a high school diploma. One or more years of college or equivalent training and strong mathematical skills are encouraged. We recommend that Level-I personnel take an approved training course and have at least 6 months of supervised training. Test quality should be reviewed by Level-II personnel or a physician, with feedback on an ongoing basis.

10.2.2 Level II: Persons with formal education (2 or more years of college-level studies in biologic sciences and/or mathematics) and training that includes 2 or more years of experience administering spirometry. One of the following credentials is recommended: Certified Respiratory Therapy Technician (CRTT), Registered Respiratory Therapist (RRT), Certified Pulmonary Function Technologist (CPFT), Registered Pulmonary Function Technologist (RPFT).

S 11.0 MONITORING:

The following should be evaluated during the performance of spirometric measurements to ascertain the validity of the results:[3,24]

11.1 acceptability of maneuver and reproducibility of FVC, FEV_1.[25]

11.2 level of effort and cooperation by the subject.

11.3 equipment function or malfunction (eg, calibration).

11.4 The final report should contain a statement about test quality.

11.5 Spirometry results should be subject to ongoing review by a supervisor, with feedback to the technologist.[12] Quality assurance and/or quality improvement programs should be designed to monitor technician competency, both initially and on an ongoing basis.

S 12.0 FREQUENCY:

The frequency with which spirometry is repeated should depend on the clinical status of the subject and the indications for performing the test.

S 13.0 INFECTION CONTROL:

Spirometry is a relatively safe procedure, but the possibility of cross-contamination exists, either from the patient-patient or the patient-technologist interface. The following guidelines should be applied whenever spirometry is performed.

13.1 Universal Precautions[26,27] should be applied in all instances in which there is the potential for exposure to blood and body fluids. The appropriate use of barriers (eg, gloves) and handwashing are recommended.[28-30]

13.2 Due to the nature of forced expiratory maneuvers and the likelihood of coughing when spirometry is performed by subjects who may have active infection with *M. tuberculosis* or other airborne organisms, the following precautions are recommended:

13.2.1 The room in which spirometry is performed should meet or exceed the recommendations of U.S. Public Health Service[31] for air changes and ventilation. The ideal situation is an area in the testing department specially ventilated for isolation patients and incorporating filtration or ultraviolet decontamination of air. If this is not possible, the patient should be returned to the isolation room as soon as possible or, alternatively, tested at the bedside using an acceptable spirometer.

13.2.2 When procedures involve patients with suspected infectious airborne diseases, barrier protection (eg, mask or personal respirator meeting regulatory agency requirements) is required.[32]

13.2.3 The use of gloves or other impermeable barriers is encouraged when contaminated equipment is handled.[3]

13.2.4 The air in a volume-displacement spirometer should be flushed out at least 5 times between subjects.[3]

13.2.5 Equipment can be reserved for the sole purpose of testing infected patients (eg, those with *M. tuberculosis* or methicillin-resistant *Staphylcoccus aureus*).

13.2.6 Infected patients can be tested at the end of the day or week and equipment can then be disassembled and disinfected.

13.2.7 Special precautions may need to be taken for immunocompromised subjects.

13.3 The mouthpiece, tubing, and any parts of the spirometer that come into direct contact with the subject should be disposable or be disinfected between patients. It is unnecessary to routinely clean the interior surface of volume-displacement spirometers.

13.3.1 The open-circuit technique (ie, no rebreathing on mouthpiece or through breathing tube) reduces the risk of infection to the patient but not to the technician.[31,32] The mouthpiece should be changed between patients, but it is not necessary to change breathing tube or hose, unless excessive water condensation occurs.[3]

13.3.2 If the closed-circuit technique (subject rebreathes on mouthpiece and through the breathing tube and spirometer) is used, the breathing tube and mouthpiece should be disposed of or disinfected between subjects.[3]

13.3.3 For flow-sensing systems in which no breathing tube is interposed between the subject and the device, inspiration from the device should be avoided or the flow-sensing element and interior tubing should be disinfected between subjects.

13.4 Bacteria filters are widely used in pulmonary function laboratories, although the need for such filters and their effectiveness is not well documented. These filters impose added resistance and have been reported to have a statistical but not clinically meaningful effect on pulmonary function test results.[33,34] If in-line filters are used during spirometry, we recommend that the equipment be calibrated with the filter installed and that the filter be discarded after use on a single subject. The use of in-line filters does not eliminate the need for regular cleaning and disinfection and does not guarantee that transmission of disease(s) cannot occur.

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AARC Clinical Practice Guideline

Selection of an Oxygen Delivery Device for Neonatal and Pediatric Patients

NPODD 1.0 PROCEDURE:

The selection of an oxygen delivery system for neonatal and pediatric patients includes patients with and without artificial airways.

NPODD 2.0 DESCRIPTION:

The administration of supplemental oxygen to neonatal and pediatric patients requires the selection of an oxygen delivery system that suits the patient's size, needs, and the therapeutic goals. Oxygen delivery systems are categorized as either low-flow, reservoir, high-flow, or enclosure systems.[1] Low-flow and reservoir systems provide an F_{DO_2} (fractional concentration of delivered oxygen) that varies with the patient's inspiratory flow and are classified as variable-performance oxygen delivery systems.[1] High-flow systems can provide a specific F_{DO_2} at flows that meet or exceed the patient's inspiratory flow requirement and are classified as fixed-performance oxygen delivery systems.[1] Enclosure systems provide a means of controlling oxygen concentration, temperature, and humidity.

2.1 Low-flow systems:

2.1.1 Nasal cannulas consist of two soft prongs that arise from oxygen supply tubing.[2] The prongs are inserted into the patient's nares, and the tubing is secured to the patient's face. Oxygen flows from the cannula into the patient's nasopharynx, which acts as an anatomic reservoir.[3] The fractional concentration of inspired oxygen (F_{IO_2}) varies with the patient's inspiratory flow.[1]

2.1.2 Nasopharyngeal catheters are soft tubes with several distal holes. The catheter should be inserted into the patient's nose to a depth equal to the distance from the ala nasi to the tragus[4] or be gently advanced and then withdrawn until it rests slightly above the uvula.[5] The tube, secured to the patient's face, is connected to oxygen supply tubing. Oxygen flows from the catheter into the patient's oropharynx, which acts as an anatomic reservoir.[5] The F_{IO_2} varies with the patient's inspiratory flow.[1]

2.1.3 Tracheostomy oxygen adapters are devices that attach either directly to a tracheostomy tube or to a heat-moisture exchanger (HME), which is then attached to the tube.[6] (HMEs recycle the heat and moisture of respired air.[6]) The oxygen supply tube connected to the adapter provides a blow-by source of oxygen that results in a variable F_{IO_2}. These devices are intended for short periods such as brief transports or to increase patient mobility.[7]

2.2 Reservoir systems:

2.2.1 Simple oxygen masks are plastic reservoirs designed to fit over the patient's nose and mouth and be secured around the patient's head by an elastic strap.[8] A reservoir effect is produced by the internal capacity of the mask.[9] Oxygen is delivered through a small-bore tube connected to the base of the mask. Holes on each side of the mask provide an egress for exhaled gases and serve as room-air entrainment ports.[8] The F_{IO_2} varies with the patient's inspiratory flow.[1]

2.2.2 Partial-rebreathing masks are similar to simple oxygen masks but contain a reservoir at the base of the mask.[8] The

reservoir receives fresh gas plus exhaled gas approximately equal to the volume of the patient's anatomic dead space.[8] The oxygen concentration of the exhaled gases combined with the supply of fresh oxygen, permits the use of flows lower than those necessary for other devices (eg, non-rebreathing masks), and potentially conserves oxygen use.

2.2.3 Non-rebreathing masks are similar to partial-rebreathing masks but do not permit the mixing of exhaled gases with the fresh gas supply.[8] A series of one-way valves placed at the reservoir opening and on the side ports, ensures a fresh oxygen supply with minimal dilution from the entrainment of room air.[8] This design provides a higher F_{IO_2} than the simple and partial-rebreathing masks and the nasal devices.[1]

2.3 High-flow systems:

2.3.1 An air-entrainment mask is designed to fit over the patient's nose and mouth and contains a short corrugated hose with a jet orifice that is connected to oxygen supply tubing.[8] Oxygen under pressure is forced through a small jet orifice entering the mask. The velocity increases causing a shearing effect distal to the jet orifice, which causes room air to be entrained into the mask.[10] The total flow provided by the mask is determined by the cross-sectional area of the entrainment ports, the diameter of the jet orifice, and the oxygen flow to the jet. The F_{DO_2} is determined by the dimensions of the jet and the entrainment ports. The entrainment mechanism is based on the principles described by Bernoulli.[10] A collar can be attached to the base of the corrugated hose for supplemental humidification,[11] and the device can be adapted to a tracheostomy collar.

2.3.2 Air-entrainment nebulizers are gas-powered, large-volume nebulizers that contain an adjustable air-entrainment port, which determines specific oxygen concentrations.[8,12] In addition to providing particulate water with or without added medication, heated nebulizers can deliver gas saturated with water vapor at body temperature.[13] A corrugated hose serves as a conduit from the nebulizer to an aerosol mask, face tent, tracheostomy collar, or T-piece.[11]

2.4 Enclosure systems:

2.4.1 Oxygen hoods are transparent enclosures designed to surround the head of the neonate or small infant.[8,14] A continuous flow of humidified oxygen is supplied to the hood.[8,14] Transparent enclosures in larger sizes (so-called tent houses or huts) are available for patients who are too big for neonatal-size hoods.[11]

2.4.2 Oxygen tents are electrically powered appliances that incorporate an air circulation system and provide temperature control.[8,14] An oxygen source is connected to a large-volume nebulizer, which provides humidification in the form of particulate water. The nebulizer is connected to a transparent canopy placed over the patient and secured under the mattress of the crib or bed. This provides an oxygen-enriched environment.[8,14]

2.4.3 Closed incubators are transparent enclosures that provide a warm environment for small infants with temperature instability.[8] Supplemental oxygen can be added to incubators but may result in an oxygen concentration.[8] The primary purpose of an incubator is to provide a temperature-controlled environment. Humidification is available through a baffled blow-over water reservoir; however, due to the high risk of infection associated with this humidification system, alternative sources are used.[8] Therefore, the incubator is not further discussed as an oxygen delivery device.[11]

NPODD 3.0 SETTING:

Oxygen delivery devices are used in a number of settings including hospitals, clinics, extended care facilities, the home, and patient transport vehicles.

NPODD 4.0 INDICATIONS:

The selection of an oxygen delivery device is indicated with:

4.1 documented hypoxemia;[15,16]
4.2 an acute situation in which hypoxemia is suspected or in which suspected regional hypoxia may respond to an increase in P_{aO_2}. Substantiation of P_{aO_2} is required within an appropriate period of time following initiation of therapy.[15,16]

NPODD 5.0 CONTRAINDICATIONS:

5.1 No specific contraindications to delivering oxygen exist when indications are judged to be present.[16]
5.2 Nasal cannulas and nasopharyngeal catheters are contraindicated in patients with nasal obstruction (eg, nasal polyps, choanal atresia).[8]
5.3 Nasopharyngeal catheters are contraindicated in the presence of maxillofacial trauma[17] and in patients in whom a basal skull fracture is present or suspected.[18]
5.4 Although opinions vary,[19-21] infants intubated for airway protection should probably be placed on CPAP (ie, physiologic CPAP) for supplemental oxygen rather than on a T-piece because of the loss of physiologic end-expiratory pressure created by an open glottis.

NPODD 6.0 HAZARDS/PRECAUTIONS/POSSIBLE COMPLICATIONS:

6.1 Physiologic:
6.1.1 The etiology of retinopathy of prematurity, especially the role of oxygen, is controversial. Care should be taken when supplemental oxygen is provided to preterm infants (< 37-weeks gestation). It is suggested that oxygen supplementation should not result in a $P_{aO_2} > 80$ torr.[22]
6.1.2 The administration of supplemental oxygen to patients with certain congenital heart lesions (eg, hypoplastic left-heart, single ventricle) may cause an increase in alveolar oxygen tension and compromise the balance between pulmonary and systemic blood flow.[23]
6.1.3 The administration of supplemental oxygen to patients suffering from paraquat poisoning or to patients receiving certain chemotherapeutic agents (eg, bleomycin) may result in pulmonary complications (eg, oxygen toxicity and pulmonary fibrosis).[24,25]
6.1.4 The administration of supplemental oxygen to patients with chronic CO_2 retention may result in a decrease in respiratory drive.[26]
6.1.5 Stimulation of the trigeminal nerves may cause alterations in respiratory pattern if the gas flow from the oxygen source is cool and is directed at the face of the infant.[27]
6.1.6 Inappropriate selection of F_{DO_2} or oxygen flow may result in hypoxemia or hyperoxemia.
6.2 Equipment-related
6.2.1 Nasal cannulas:
6.2.1.1 Skin irritation can result from material used to secure the cannula[3,28] or from local allergic reaction to polyvinyl chloride.[29]
6.2.1.2 Improper sizing can lead to nasal obstruction or irritation.[3,30]
6.2.1.3 Displacement can lead to loss of oxygen delivery.[3,30]
6.2.1.4 Inadvertent CPAP may be administered depending upon the size of the nasal cannula, the gas flow, and the infant's anatomy.[31]
6.2.1.5 Irritation can result if flows are excessive.
6.2.2 Nasopharyngeal catheters:
6.2.2.1 Improper insertion can cause gagging[5] and nasal or pharyngeal trauma.[5]
6.2.2.2 Excessive flow can produce pain in the frontal sinuses.[8]
6.2.2.3 Pneumocephalus is a rare but possible complication.[32]
6.2.2.4 Excessive secretions and/or mucosal inflammation can result.[5]
6.2.2.5 Skin irritation may result from material used to secure the cannula[3,28] and/or from local allergic reaction to polyvinyl chloride.[29]
6.2.2.6 Occlusion of distal openings may occur.[4]
6.2.2.7 Excessive flow may cause gastric distention.[4]
6.2.3 Masks:
6.2.3.1 Aspiration of vomitus may be more likely when a mask is in place.

6.2.3.2 Irritation may result from tight application.[8]

6.2.3.3 Rebreathing of CO_2 may occur if total O_2 flow is inadequate.[12,33]

6.2.4 Air-entrainment nebulizers:

6.2.4.1 produce high noise levels in enclosed environments (eg, hoods, incubators) and may induce hearing impairment;[34] when an air-entrainment nebulizer is used in an enclosed environment, the entrainment port should be set on 100% (ie, closed) and the nebulizer powered either by a blender or by compressed air with titration of oxygen to the desired concentration.[11]

6.2.4.2 are susceptible to contamination;[35-37]

6.2.4.3 may cause bronchoreactivity in patients with reactive airways when used with nonisotonic solutions;[38-40]

6.2.4.4 may create unwanted torque and increase the likelihood of inadvertent extubation or decannulation of the patient when used with a T-piece and applied directly to an endotracheal or tracheostomy tube;[11]

6.2.4.5 may not provide particles of desired size range and in a predictable dose;[41]

6.2.4.6 if unheated, may induce cold stress in neonates;[27,42]

6.2.4.7 Condensate in tubing may result in advertent lavage when attached to the endotracheal tube.

6.2.5 Hoods and transparent enclosures:

6.2.5.1 Prolonged exposure to humidified oxygen may increase risk for cutaneous fungal infection.[43]

6.2.5.2 Inadequate or loss of gas flow may result in hypoxia or hypercapnia.

6.2.5.3 Temperature within enclosures should be closely monitored to reduce the potential for cold stress or apnea from overheating in neonates.[27]

6.2.5.4 Use of an improperly sized hood can result in irritation of the infant's skin.[11]

6.2.6 Oxygen tents:

6.2.6.1 Potential exists for electric shock or fire from electrical or battery-operated devices inside the tent.[8,11]

6.2.6.2 Potential exists for electric shock from static electrical discharge from plastic tent canopies.[8,11]

6.2.6.3 Large-volume nebulizers used to supply oxygen to tents are susceptible to contamination.[35-37]

6.2.6.4 Loss of the air circulation system may result in failure to cool the tent.[11]

6.2.6.5 Potential exists for asphyxiation if the patient becomes lodged between the mattress and the tent.

6.2.7 Tracheostomy oxygen adapters: Adapters may create unwanted torque and increase the likelihood of inadvertent decannulation of the patient, and HMEs may increase work of breathing to an unacceptable level in patients < 8 kg if dead space and resistance are high.[44]

6.3 During laser bronchoscopy, minimal levels of supplemental oxygen should be used to decrease the risk of intratracheal ignition.[16,45]

6.4 Fire hazard is increased in the presence of increased oxygen concentrations.[16]

6.5 Bacterial contamination has been associated with certain nebulization and humidification systems.[35-37]

NPODD 7.0 LIMITATIONS:

7.1 Nasal cannulas:

7.1.1 Changes in minute ventilation and inspiratory flow affect air entrainment and result in fluctuations in F_{IO_2}.[46,47]

7.1.2 Prongs are difficult to keep in position, particularly with small infants.[3,28,48]

7.1.3 The effect of mouth versus nose breathing on F_{IO_2} remains controversial.[49-52]

7.1.4 Use may be limited by the presence of excessive mucus drainage, mucosal edema, or a deviated septum.[8]

7.1.5 Maximum flow should be limited to 2 L/min in infants and newborns.[46,47,53]

7.1.6 Care should be taken to keep the cannula tubing and straps away from the neck to prevent airway obstruction in infants.

7.1.7 Discrepancies between set and delivered flow can occur in the same flowmeter at different settings and among different flowmeters.

7.1.8 Discrepancies in flow and oxygen concentration between set and delivered values can occur in low-flow blenders at flows below the recommended range of the blender.

7.2 Nasopharyngeal catheters:

7.2.1 Method is in less common use because of the complexity of care.

7.2.2 F_{IO_2} is difficult to control and measure.[46,47]

7.2.3 Effect of mouth versus nose breathing on F_{IO_2} remains controversial.[48-52]

7.2.4 Use may be limited by excessive mucus drainage, mucosal edema, or the presence of a deviated septum.[5]

7.2.5 Catheter should be cleared frequently to prevent occlusion of the distal holes.[4,5] The patient should be observed for evidence of catheter occlusion, and the catheter should be alternated between nares every 8-12 hours and changed daily.[30]

7.2.6 Catheter sizes less than 8 Fr are less effective in oxygen delivery.[4]

7.2.7 Lower oxygen concentrations are delivered if the catheter is placed in the nose rather than in the pharynx.[4]

7.2.8 Low-flow flowmeters (< 3 L/min) should be used.[4]

7.2.9 Discrepancies between set and delivered flow can occur in the same flowmeter at different settings and among different flowmeters.

7.2.10 Discrepancies in flow and oxygen concentration between set and delivered values can occur in low-flow blenders at flows below those recommended by the manufacturer.

7.3 Masks:

7.3.1 provide variable F_{IO_2} depending on inspiratory flow and construction of the mask's reservoir[11] and are not recommended when precise concentrations are required;[15,54]

7.3.2 are confining and may not be well tolerated;[55]

7.3.3 interfere with feeding;[5]

7.3.4 may not be available in sizes appropriate for all patients;

7.3.5 require a minimum flow to avoid possible rebreathing of CO_2.[12,33]

7.3.6 The maximum F_{IO_2} attainable with a simple, non-rebreathing or partial-rebreathing mask in neonates, infants, and children has not been well documented.[33]

7.3.7 The performance of air-entrainment masks may be altered by resistance to flow distal to the restricted orifice (resulting in higher F_{DO_2} and lower total flow delivered). The total flow from air-entrainment masks at settings greater than 0.40 may not equal or exceed the patient's inspiratory flow.[8,16]

7.3.8 Performance is altered if the entrainment ports are blocked.

7.4 Air-entrainment nebulizers:

7.4.1 are vulnerable to alterations described in Section 7.3.7;

7.4.2 should have temperature monitored if they are heated (Cool mist is not recommended for newborns because of the potential for cold stress.[27,42] In newborns, the temperature of the gas-aerosol mixture at the patient should be approximately equal to the desired environmental temperature.[42]);

7.4.3 may have performance altered by resistance to flow distal to the restricted orifice (resulting in higher F_{DO_2} and lower total flow delivered). The total flow from air-entrainment nebulizers at settings greater than 0.40 may fail to equal or exceed the patient's inspiratory flow.[8,16] However, increasing the oxygen flow to the inlet of the nebulizer may produce a higher delivered total flow.

7.5 Hoods and tents:

7.5.1 O_2 concentrations may vary within the hood or tent.[8,11,16] O_2 concentrations should be measured as near the nose and mouth as possible.[11] Opening any enclosure decreases the O_2 concentration. For infants and children confined to tents, nasal O_2 may need to be supplied during feeding and nursing care. Flows $\geq$ 7 L/min are required to wash out carbon dioxide.[11,12]

7.5.2 Devices can be confining and isolating. Dense fog or excessive condensation can develop, which may frighten the child and limit observation.[11]

7.5.3 Only low-to-moderate concentrations < 0.40 can be achieved in a tent.[11]

7.5.4 Concentration in a hood can be varied from 0.21 to 1.0 and is more stable than in a tent.[9]

7.5.5 Temperature of the gases in the hood should be maintained to provide a neutral thermal environment.[8,16,42]

7.5.6 High gas flows may produce harmful noise levels.[34,56]

7.6 Tracheostomy oxygen adapters provide variable F_{IO_2}s. HMEs should have minimum dead-space volume especially when used with neonates.[44,57,58] Resistance within an HMEs can increase when water is absorbed by the hygroscopic inserts[59] or when secretions are coughed into the device.[60]

NPODD 8.0 ASSESSMENT OF NEED:

Need is determined by measurement of inadequate oxygen tensions and saturations by invasive or noninvasive methods[61,62] and/or the presence of clinical indicators as previously described.[16] Supplemental oxygen flow should be titrated to maintain adequate oxygen saturation as indicated by pulse oximetry (S_{pO_2}).

8.1 Nasal cannulas and nasopharyngeal catheters are used when the need exists to

8.1.1 provide low-level supplemental oxygen to the infant or child;[46]

8.1.2 feed the infant without interrupting oxygen delivery;[5,63,64]

8.1.3 increase mobility.[5,63,64]

8.2 Simple oxygen masks are used to provide supplemental O_2 in the moderate range (0.35-0.50, depending on size and minute ventilation) for short periods of time (eg, during procedures, for transport, in emergency situations).[12,15,16,54]

8.3 Partial rebreathing masks are used to conserve the oxygen supply when higher concentrations (F_{IO_2} > 0.4, < 0.6) are warranted (eg, during transport).[15,16,50,54]

8.4 Non-rebreathing masks are used to deliver concentrations ≥ 0.60 or specific concentrations (as from a blender).[15,16,54]

8.5 Air-entrainment masks provide a flow of gas of predetermined precise oxygen concentration (24-40%)[10,46,65,66] that exceeds the patient's inspiratory flow. At the 50% setting, the total flow from the device may not meet the inspiratory flow.[16,54,65,66]

8.6 Air-entrainment nebulizers, although not recommended, can be used when high levels of humidity or aerosol are desired (as with a bypassed upper airway).[40,67] The patient application device can be a tracheostomy collar, face tent, aerosol mask, or blow-by arrangement.[11,16,55]

8.7 Hoods are used to provide

8.7.1 controlled F_{IO_2} in infant and small children;[8]

8.7.2 controlled F_{IO_2} and/or increased heated humidity to patients who cannot tolerate other devices;

8.7.3 controlled F_{IO_2} when the chest, abdomen, and extremities must be accessible to caregivers;[8]

8.7.4 the oxygen concentrations necessary for oxygen challenge (hyperoxia) tests in the spontaneously breathing neonate.

8.8 Tents are appropriate to deliver supplemental oxygen and/or cool, high-humidity gas mixtures to pediatric patients:

8.8.1 with laryngotracheobronchitis.[68,69]

8.8.2 with artificial airways in whom direct attachment of a device to the airway is contraindicated.[68]

8.8.3 who are too big for hoods.[8]

8.8.4 with the need for comfort, without compromising therapy.[69]

8.9 Tracheostomy oxygen adapters, which may or may not be coupled with HMEs, are used to deliver oxygen to a tracheostomy.[44]

NPODD 9.0 ASSESSMENT OF OUTCOME:

Outcome is assessed by determining whether the device selected produces an appropriate increase in oxygen saturation, proves to be appropriate for the patient, allows adequate patient monitoring, and facilitates patient care.

NPODD 10.0 RESOURCES:

10.1 Equipment

10.1.1 Oxygen source:

10.1.1.1 Cylinder—must meet Department of Transportation (DOT) standards, Compressed Gas Association

(CGA) standards, and National Fire Protection Association (NFPA) recommendations, and appropriate regulator and wrenches must be supplied;

10.1.1.2 Concentrators (or enrichers).[70,71]

10.1.1.3 Bulk supplies should meet NFPA standards.

10.1.2 Delivery accessory equipment:

10.1.2.1 oxygen tubing;

10.1.2.2 corrugated aerosol tubing and water trap.

10.1.3 Humidifiers—No subjective or objective evidence supports routine humidification of O_2 at flows ≤ 4 L/min.[15,70] However, it is not known whether the use of a bubble humidifier with a nasal cannula in the neonate has benefit and the use of a bubble humidifier can verify oxygen delivery at flows < 1 L/min. HMEs with low dead space are appropriate for short-term use in patients with artificial airways.

10.1.4 Blenders—Although blenders have been used in weaning neonates with a nasal cannula from oxygen, it appears that using a very-low flowmeter (0-200 mL) may be more reliable.[72]

10.1.5 Compensated, low-range flowmeters[73] adjustable in increments < 0.125 L/min.[4,47,74]

10.1.6 Oxygen analyzers—Physical and electrical O_2 analyzers are suitable for intermittent sampling, and electrochemical analyzers are suitable for continuous sampling.[75]

10.1.7 Noninvasive oxygen monitors—transcutaneous (TcO_2) monitor[61,76] or pulse oximeter.[62,77]

10.1.8 Nebulizer solutions—sterile water or sterile normal saline solution.

10.2 Personnel:

10.2.1 Health care providers responsible for delivery of oxygen should have demonstrated and documented knowledge and skills related to:

10.2.1.1 oxygen delivery systems and their limitations;

10.2.1.2 assembly, care, and use of oxygen delivery systems;

10.2.1.3 performance of the necessary subjective and objective assessments in order to determine effectiveness of oxygen therapy;

10.2.1.4 clinical assessment skills to recommend changes in oxygen therapy;

10.2.1.5 provision of comprehensive patient and lay caregiver instruction.

10.2.2 When supplemental oxygen is to be used out of the hospital setting, the patient and/or family member or lay caregiver should:

10.2.2.1 demonstrate proper use and understanding of oxygen delivery device;

10.2.2.2 demonstrate proper assembly, care, and cleaning of oxygen delivery device;

10.2.2.3 demonstrate an understanding of how, when, and what to report to a physician or surrogate.

NPODD 11.0 MONITORING:

11.1 Patient:

11.1.1 clinical assessment including but not limited to cardiac, pulmonary, and neurologic status and apparent work of breathing;

11.1.2 assessment of physiologic variables: noninvasive or invasive measurement of oxygen tensions or saturation in any patient treated with oxygen—within 1 hour of initiation for the neonate.[16,22]

11.2 Equipment:

11.2.1 All oxygen delivery systems should be checked at least once each day.[16] More frequent checks by calibrated analyzer are necessary in systems:

11.2.2.1 susceptible to variation in oxygen concentration;[16]

11.2.2.2 applied to patient's with artificial airways;[16]

11.2.2.3 continuous analysis is recommended in hoods.[78]

11.2.2.4 oxygen should be analyzed close to the infant's face.

11.2.3 All heated delivery systems should be continuously monitored for temperature.

NPODD 12.0 FREQUENCY:

12.1 Selection of a device is made at the initia-

tion of therapy, after careful assessment of need and patient characteristics.

12.2 The change from one type of device to another is based on a change in the patient's condition or ability to use a specific device. (Oxygen therapy should be administered continuously unless the need has been shown to be associated only with specific situations eg, exercise, feeding, or other stress.[15,79,80])

NPODD 13.0 INFECTION CONTROL:

13.1 Universal Precautions and measures to limit the transmission of tuberculosis must be adhered to at all times.[81,82]

13.2 Low-flow systems

13.2.1 Under normal circumstances, low-flow oxygen systems do not present clinically important risk of infection and do not require routine replacement on the same patient.[15,16,83]

13.2.2 Nasopharyngeal catheters should be changed every 24 hours.[5]

13.3 Reservoir systems—Under normal circumstances, reservoir systems as defined for this guideline do not present clinically important risk of infection and do not require routine replacement on the same patient.

13.4 High-flow systems

13.4.1 Large-volume nebulizers should be changed every 24 hours when applied to patient's with an artificial airway.[83]

13.4.2 In the absence of definitive studies to support change-out intervals on nonintubated patients, results of institution-specific and patient-specific surveillance measures should dictate the frequency with which such equipment is replaced.

13.5 Enclosure systems—There is no recommendation regarding the frequency of changing mist-tent nebulizers and reservoirs while in use on the same patient,[83] but mist-tent nebulizers are probably as prone to contamination as any other nebulizer and we recommend that they be handled in the same manner.

13.6 Other devices—Between patients, subject equipment (eg, probes, oxygen sensors) to high level disinfection.[83]

13.7 Nebulizer solutions—Use only sterile fluids and dispense them aseptically.[83]

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AARC Clinical Practice Guideline

Selection of a Device for Delivery of Aerosol to the Lung Parenchyma

DALP 1.0 PROCEDURE:

Selection of a device for delivery of aerosol to the lung parenchyma

DALP 2.0 DESCRIPTION:

A device selected for administration of pharmacologically active aerosol to the lung parenchyma should produce particle sizes with a mass median aerodynamic diameter (MMAD) of 1-3 μ.[1-4]

Such devices include ultrasonic nebulizers (USNs, including the Porta-Sonic[5,6]), some large volume nebulizers (LVNs, such as the Small Particle Aerosol Generator,™ or SPAG, which is intended only for ribavirin delivery[7]), some small volume nebulizers (such as the Circulaire,[8] RespirGard II,[5,6,9] and Pari IS 2,[6]) Other devices including metered dose inhalers (MDIs) may be developed for parenchymal deposition. Although some parenchymal deposition of particles < 1 μ probably does occur, selection of a specific device should be based on its ability to produce particles within MMAD of 1-3 μ.

DALP 3.0 SETTING:

Aerosol therapy can be administered in a number of settings including but not limited to hospital, clinic, extended care facility, or home.

DALP 4.0 INDICATIONS:

The indication for selecting a suitable device is the need to deliver a topical medication (in aerosol form) that has its site of action in the lung parenchyma or is intended for systemic absorption. Such medications may possibly include antibiotics, antivirals, antifungals, surfactants, and enzymes.

DALP 5.0 CONTRAINDICATIONS:

5.1 No contraindications exist to choosing an appropriate device for parenchymal deposition.
5.2 Contraindications related to the substances being delivered may exist. Consult the package insert for product-specific contraindications to medication delivery.

DALP 6.0 HAZARDS/COMPLICATIONS:

6.1 Malfunction of device and/or improper technique may result in underdosing or overdosing.
6.2 In mechanically ventilated patients, the nebulizer design and characteristics of the medication may affect ventilator function (eg, filter obstruction, altered tidal volume, decreased trigger sensitivity) and medication deposition.[10,11]
6.3 Complications related to specific pharmacologic agents can occur.
6.4 Aerosols may cause bronchospasm or irritation of the airway.
6.5 Exposure to medications[12-23] and patient-generated droplet nuclei may be hazardous to clinicians.[24]

6.5.1 Exposure to medication should be limited to the patient for whom it has been ordered. Nebulized medication that is released into the atmosphere from the nebulizer or exhaled by the patient becomes a form of "secondhand" exposure that may affect health-care providers and others in the vicinity of the treatment.

There has been increased awareness of possible health effects of aerosols, such as ribavirin and pentamidine. Anecdotal reports associate symptoms such as conjunctivitis, decreased tolerance to presence of contact

lenses, headaches, bronchospasm, shortness of breath, and rashes in health-care workers exposed to secondhand aerosols. Similar concerns have been expressed concerning health-care workers who are pregnant or are planning to be pregnant within 8 weeks of administration. Less often discussed are the potential exposure effects of aerosolized antibiotics (which may contribute to the development of resistant organisms), steroids, and bronchodilators.[25]

Because the data regarding adverse health effects on the health-care worker and on those casually exposed are incomplete, the prudent course is to minimize exposure in all situations.[26]

6.5.2 The Centers for Disease Control and Prevention recommend addressing exposure control issues by (1) administrative policy, (2) engineering controls, and (3) personal protective equipment, in that order.[27,28]

6.5.2.1 Administrative controls:
Should include warning signs to apprise all who enter a treatment area of potential hazards of exposure. Accidental exposures should be documented and reported according to accepted standards.

Measures to reduce aerosol contamination of room air include:

6.5.2.1.1 discontinuing nebulization of medication while patient is not breathing the aerosol;

6.5.2.1.2 ensuring that staff who administer medications understand risks inherent with the medication and procedures for safely disposing of hazardous wastes;

6.5.2.1.3 screening of staff for adverse effects of exposure to aerosol medication;

6.5.2.1.4 providing alternative assignments for those staff who are at high risk of adverse effects from exposure (eg, pregnant women or those with demonstrated sensitivity to the specific agent).

6.5.2.2 Engineering controls:

6.5.2.2.1 Filters or filtered scavenger systems to remove aerosols that cannot be contained.

6.5.2.2.2 Frequent air exchanges to dilute concentration of aerosol in room to eliminate 99% of aerosol before the next patient enters and receives treatment in the area.

6.5.2.2.3 Booths or stalls for sputum induction and aerosolized medication administration in areas in which multiple patients are treated. Booths or stalls should be designed to provide adequate air flow to draw aerosol and droplet nuclei from the patient and into an appropriate filtration system, with exhaust directed to an appropriate outside vent.

6.5.2.2.4 Handling of filters, nebulizers, and other contaminated components of the aerosol delivery system used with suspect agents (such as pentamidine and ribavirin) as hazardous waste.

6.5.2.3 Personal protection devices:

6.5.2.3.1 Personal protection devices should be used to reduce exposure when engineering alternatives are not in place or are not adequate. Use properly fitted respirators with adequate filtration when exhaust flow cannot adequately remove aerosol particles.[28]

6.5.2.3.2 Goggles, gloves, and gowns should be used as splatter shields and to reduce exposure to medication residues and body substances.

DALP 7.0 LIMITATIONS OF PROCEDURE OR DEVICE:

7.1 A relatively small fraction of nebulizer output deposits in the lung parenchyma.[2]

7.2 Efficacy of the device is technique dependent (eg, coordination, ability to follow instructions, and breathing pattern including inspiratory flow and inspiratory hold).[2,29-32]

7.3 Efficacy of the device is design dependent (ie, output and particle size).[33-36]

7.4 The following clinical situations are associated with reduced deposition of aerosol to the lung parenchyma and may require consideration of increased dose:

7.4.1 Mechanical ventilation[37-42]

7.4.2 Artificial airways[43-45]

7.4.3 Reduced airway caliber (eg, infants and pediatrics)[46]

7.4.4 Severity of obstruction[4]

7.4.5 Hydrophilic formulations[2]

7.4.6 Failure of patient to comply with procedure.

7.5 Limitation of specific devices:

7.5.1 MDI:

7.5.1.1 Particle size varies with drug formulation, propellants, evaporation rate, and humidity.

7.5.1.2 Accessory device is recommended with MDI use[26,47]

7.5.1.2.1 assure optimal deposition

7.5.1.2.2 reduce oropharyngeal deposition

7.5.1.2.3 decrease caregiver exposure.

7.5.1.3 Relatively few formulations are targeted to the lung parenchyma.

7.5.2 SPAG™[40] (intended only for ribavirin delivery)

7.5.2.1 Limited to acute and critical care setting

7.5.2.2 Requires close monitoring

7.5.2.3 Requires compressed gas source

7.5.2.4 Vulnerable to contamination

7.5.2.5 Reconcentration of solution may occur over a long period of time due to evaporation by dry gas source.[49-52]

7.5.3 SVN:

7.5.3.1 Few devices produce particles with MMAD of 1-3 μ.

7.5.3.2 MMAD may vary with nebulizer model, medication, gas flow, and gas pressure.

7.5.3.3 Requires compressed gas source.

7.5.3.4 Vulnerable to contamination.

7.5.3.5 Reconcentration may occur.[49-52]

7.5.4 LVN:

7.5.4.1 Generally limited to acute and critical care setting although anecdote suggest some home use.

7.5.4.2 Requires close monitoring.

7.5.4.3 Few devices produce particles with MMAD of 1-3 μ.

7.5.4.4 Vulnerable to contamination.

7.5.5 USN:

7.5.5.1 Cost may be a factor.

7.5.5.2 May be mechanically unreliable.

7.5.5.3 Requires electrical power source.

7.5.5.4 Some units do not reliably produce MMAD of 1-3 μ.[44-54]

7.5.5.5 Reconcentration of solution may occur over long period of time due to evaporation by heat generated by piezoelectric cell.[53,55] The ultrasonic device may denature the medication.[56,57]

DALP 8.0 ASSESSMENT OF NEED (Selection Criteria for Device):

8.1 Availability of prescribed drug in solution or MDI formulation.

8.2 Availability of appropriate scavenging or filtration equipment.

8.3 Patient preference for a given device that meets therapeutic objectives.

8.4 Although specific devices may give known ranges of particle size and output, clear superiority of any one method or device for achieving specific clinical outcomes has not been established. Cost, convenience, effectiveness, and patient tolerance of procedure should be considered.[26,53]

8.5 When spontaneous ventilation is inadequate (eg, kyphoscoliosis, neuromuscular disorders, or respiratory failure) consider augmentation with mechanical ventilation.

DALP 9.0 ASSESSMENT OF OUTCOME

Appropriate device selection is reflected by evidence of

9.1 use of proper technique in applying device;

9.2 patient compliance with procedure;

9.3 a positive clinical outcome. (However, appropriate device selection and application does not guarantee a positive outcome.)

DALP 10.0 RESOURCES

10.1 Equipment:

10.1.1 MDI and accessory device (holding chamber or spacer).
10.1.2 SPAG (nebulizer with drying chamber supplied by manufacturer); gas source; tent, hood or ventilator; scavenger or filter system to prevent aerosol from being released outside immediate treatment area.
10.1.3 SVN, gas source, tubing, one-way valves, mouthpiece or mask, scavenger or filter system to prevent aerosol from being released outside immediate treatment area.
10.1.4 LVN, gas source, flowmeter, connecting tubing, mouthpiece or mask scavenger or filter system to prevent aerosol from being released outside immediate treatment area.
10.1.5 Mechanical ventilator with SPAG unit, adapter in inspiratory line of circuit, filter system in expiratory line to prevent obstruction of expiratory valves, scavenger or filter system to prevent aerosol from being released outside immediate treatment area.
10.1.6 IPPB machine (ie, pressure-cycled ventilator), nebulizer gas source, connecting tubing, mouthpiece or mask, scavenger or filter system to prevent aerosol from being released outside immediate treatment area.

10.2 Personnel

10.2.1 Level-II personnel should establish the need for a specific device by patient assessment and complete the first administration of the medication. Level-II personnel should continue to care for the unstable patient. We recommend that the Level-II caregiver be credentialed (eg, RRT, CRTT, RN). Level-II personnel must have documented knowledge of and demonstrated ability to perform regarding:

10.2.1.1 Indications and limitations for SPAG, MDI with accessory devices, SVN, LVN, and USN.
10.2.1.2 Proper use, maintenance and cleaning of equipment, including filter and scavenging systems.
10.2.1.3 Risks inherent to the medications and specific devices.
10.2.1.4 Safe disposal of hazardous wastes and medical waste.
10.2.1.5 Optimal breathing patterns and coughing techniques.
10.2.1.6 Technique modification in response to adverse reactions.
10.2.1.7 Dosages and/or frequency modification as prescribed, in response to severity of symptoms.
10.2.1.8 Assessment of patient condition and response to therapy.
10.2.1.9 Performance of auscultation, inspection, and monitoring of vital signs.
10.2.1.10 Performance of peak expiratory flowrate, spirometry, and ventilatory mechanics.
10.2.1.11 Recognition and response to therapeutic or adverse response and complications of medication administration and/or the procedure.
10.2.1.12 Understanding and compliance with Universal Precautions[58] and guidelines for prevention of the spread of tuberculosis[28,59] and other infection control measures.

10.2.2 Level-I caregiver may be the provider of service to the stable patient after Level-II personnel have established need for a specific device by patient assessment and the first administration has been completed. We recommend that Level-I personnel be credentialed (eg, RRT, CRTT, RN). They must have documented knowledge and demonstrated ability to perform related to:

10.2.2.1 preparing, measuring, and mixing medication;
10.2.2.2 demonstrating proper technique for administration of medication;
10.2.2.3 using equipment properly;
10.2.2.4 cleaning equipment;
10.2.2.5 properly disposing of wastes;
10.2.2.6 encouraging effective breathing patterns and coughing techniques;
10.2.2.7 modifying technique in response to adverse reactions as instructed, with appropriate communication with physician, in response to severity of symptoms;

10.2.2.8 implementing and observing Universal Precautions and guidelines to avoid transmission of tuberculosis and proper infection control.

DALP 11.0 MONITORING:

11.1 Performance of the device and scavenging system
11.2 Technique of device application
11.3 Assessment of patient response

DALP 12.0 FREQUENCY:

A device for delivery of aerosolized medication to the lung parenchyma is selected

12.1 on initiation of therapy, after careful assessment of need (as outlined);
12.2 when the decision is made to change the device because of a change in patient condition or because the patient is unable to use the specific device.

DALP 13.0 INFECTION CONTROL:

13.1 Caregiver should exercise Universal Precautions for body substance isolation and follow CDC recommendations for control of exposure to tuberculosis and droplet nuclei.[28,58]
13.2 Nebulizers should not be used between patients without disinfection.
13.3 Nebulizers should be changed or sterilized:

13.3.1 at conclusion of dose administration or at least every 24 hours;
13.3.2 at 24-hour intervals with continuous administration;
13.3.3 when visibly soiled.

13.4 Nebulizers should not be rinsed with tap water between treatments but may be rinsed with sterile water. CDC Guidelines recommend sterile water rinsing and air drying.[60]
13.5 All medications should be handled aseptically.
13.6 CDC guidelines suggest that if multidose medications containers are dated upon opening and handled aseptically, medication may be used until exhausted or until the manufacturer's expiration date unless visible or suspected contamination occurs.[61,62] However, it is not clear whether this applies only to solutions containing a bacteriostatic agent. Caution is advised particularly for reconstituted solutions.

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AARC Clinical Practice Guideline

Training the Health-Care Professional for the Role of Patient and Caregiver Educator

THCP 1.0 PROCEDURE:

The process of enabling the health-care provider (HCP) to facilitate the patient and/or caregiver's acquisition of knowledge, skills, understanding, and positive attitudes related to the patient's medical condition and its management.

THCP 2.0 DESCRIPTION/DEFINITION:

2.1 The process of training the HCP as patient and/or caregiver educator includes addressing and assuring adequate knowledge, skills, and attitude mastery for patient rapport and affective teaching.[1] (HCP is defined as any health-care professional involved in the care of the patient).

2.2 The ultimate goal of the process is to provide education to the patient and/or caregiver that equips him/her with the knowledge, skills, and attitudes to better understand the patient's condition and to more fully participate in health care. (The caregiver is defined as any person who plays a significant role in the patient's life. This includes individuals who may not be legally related to the patient). This allows a cooperative effort between the health-care provider and patient.[1,2]

THCP 3.0 SETTING:

Any location in which HCPs participate in or can be prepared for patient and/or caregiver education and in which resources are available, including, but not limited to:

3.1 acute care hospitals;
3.2 outpatient treatment or rehabilitation centers;
3.3 physicians' offices;
3.4 extended care or skilled nursing facilities or the patient's home;
3.5 educational seminars, workshops, and symposia;
3.6 postsecondary technical schools, colleges, and universities.

THCP 4.0 INDICATIONS

4.1 The presence of HCPs who must participate in education of patients and caregivers who need:
4.1.1 knowledge concerning condition or care;[1]
4.1.2 skills to participate effectively in care;[1]
4.1.3 proper attitude or motivation to participate effectively in care.[3]

4.2 Evidence that the knowledge, skills, or attitudes of HCPs are inadequate to convey the necessary information or demonstrate skills to patients, including evidence of:
4.2.1 lack of knowledge of effective educational principles and practices specific to patient's or caregiver's age and development;[2,4-9]
4.2.2 inability to accurately assess patient's or caregiver's educational needs;[2,7]
4.2.3 inability to prepare educational objectives and establish instructional goals;[1,7,10]
4.2.4 lack of knowledge of education methods to accomplish objectives and meet goals;
4.2.5 inability to prepare patient or caregiver education materials;[11-13]
4.2.6 inability to offer supervised practice of skills;

4.2.7 inability to assess outcomes of patient or caregiver education;[2]
4.2.8 inability to appropriately offer feedback to patient or caregiver;[14,15]
4.2.9 inability to modify educational efforts as the result of learning outcome assessment and feedback from patients.[5,6,10]

THCP 5.0 CONTRAINDICATIONS:

There are no contraindications to HCP training to offer patient and/or caregiver education when a need exists.

THCP 6.0 HAZARDS/COMPLICATIONS:

Inadequate training of the HCP can lead to harm to the patient or result in the patient's inability to participate in his/her health-care management.

THCP 7.0 LIMITATIONS OF METHOD:

The effectiveness of educating the HCP to offer patient and/or caregiver education may be limited by:

7.1 Limitations in the HCP:
7.1.1 unreceptive or incapable HCP;[2,6,7]
7.1.2 lack of educational preparation;[2,7]
7.1.3 absence of interdisciplinary cooperation;[2]
7.1.4 inability to deal with age or development-specific learning patterns;[2]
7.1.5 inadequate knowledge of cultural or religious practice that may affect educational process;[2]
7.1.6 inability of HCP to communicate effectively.
7.2 System limitations:
7.2.1 inadequate time to prepare the HCP due to other patient care responsibilities;
7.2.2 inadequate space, energy, or financial resources;
7.2.3 insufficient or unavailable faculty and preceptors for HCP training program;
7.2.4 inconsistency in information provided to the HCP;
7.3 Limitations in the patient or caregiver:
7.3.1 absolute refusal to participate in education;
7.3.2 lack of positive attitude or motivation to participate;[5,6,16]
7.3.3 lack of basic education necessary to benefit from instruction;[5,8,10]
7.3.4 perception of cultural conflict or presence of a language barrier.[2]

THCP 8.0 ASSESSMENT OF NEED

All HCPs who have patient care contact and opportunity to provide patient and/or caregiver education should be assessed for training as education providers[2] by

8.1 observation of HCP in patient education setting to determine if needed skills are present;
8.2 verbal questioning by specialist as to knowledge of topics being taught.

THCP 9.0 ASSESSMENT OF OUTCOME:

9.1 Evidence by HCP evaluations that the training has met the goals and objectives for preparing patient and/or caregiver educators:
9.1.1 verbal or written evaluation;
9.1.2 observation of HCP in teaching setting.
9.2 The desired outcome of training the HCP as a provider of patient and/or caregiver education is also assessed by meeting the goals of patient and/or caregiver education:
9.2.1 knowledge gained by the patient or patient care provider;[10]
9.2.2 skills mastered by the patient or the patient care provider;[2,14]
9.2.3 positive change in patient outlook and/or attitude;[6]
9.2.4 compliance of patient or caregiver in following the care plan.
9.3 Long-term assessment through departmental or institutional continuous quality improvement indicators or other quality monitoring system.

THCP 10.0 RESOURCES:

10.1 Written materials
10.2 Audiovisual materials
10.3 Anatomic models, mannequins, patient care equipment
10.4 Computer and software

10.5 "Appendix—Essentials of the Teaching Process" in Clinical Practice Guideline "Providing Patient and Caregiver Education"[17]

10.6 Personnel

10.6.1 Education specialists with training in patient education

10.6.2 Training specialists in general adult education

10.7 Education seminars and clinical seminars with emphasis in patient education

10.8 Formal 'for-credit' patient and adult education courses provided by postsecondary institutions of learning

THCP 11.0 MONITORING

11.1 The monitoring of the process of training the HCP as educator of the patient and/or patient care provider should include evidence of:

11.1.1 classes and inservice training for HCPs as educators;

11.1.2 availability of written and audiovisual materials, computers, and software for use with computers for training HCP as educators;

11.1.3 HCP evaluation of training effectiveness.

11.2 Patient and caregiver education directed at meeting the goals and objectives of the education plan

THCP 12.0 FREQUENCY:

12.1 Spontaneous: as often as the HCP has contact with patients or caregivers requiring education for which the HCP is not prepared. (This should take place outside of patient care area.)

12.2 Scheduled: a formal HCP-as-educator training program. (This may be included in orientation or as a skills laboratory at least once per year.)

12.3 As available: training programs offered by external sources such as professional associations and accrediting agencies.

THCP 13.0 INFECTION CONTROL:

The presence of transmissible infection and/or the presence of compromised immunity in the patient should be taken into consideration when patient and/or caregiver education is undertaken. Appropriate steps to protect patient, caregiver, health-care provider, and trainer should be included in the education plan.

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AARC Clinical Practice Guideline

Providing Patient and Caregiver Training

PCGT 1.0 PROCEDURE

A health-care provider-initiated process to facilitate the patient or caregiver's acquisition of knowledge and skills related to his or her medical condition and participation in its management.

PCGT 2.0 DESCRIPTION/DEFINITION

2.1 Patient and caregiver education provides the patient and family with the means of participating in the patient's health-care management to the extent feasible, depending on physical condition and awareness. (The term family encompasses the person(s) who play a significant role in the patient's life and may include persons who are not legally related to the patient.)[1-3]

2.2 The training process should occur with every encounter between the health-care provider (HCP) and the patient. (The health-care provider is defined as any health-care professional involved in the care of the patient.)

2.3 The goal of the HCP should be to elicit a positive change in the patient's behavior through the use of verbal, written, and visual communication in the affective, cognitive, and psychomotor domains. Coordinated efforts by HCPs should provide the patient with an improved understanding of health-care needs, therapy, and the importance of adherence to medical regimen and candid communication with caregivers. This should enable the patient to better manage the disease through cooperation with the caregiver and HCP in an active partnership.[2,3] All members of the team need to be aware of these goals as an aspect of the patient's total care.

2.4 A final goal of the HCP is to provide the patient and family with the means to reap the economic benefits of improved utilization of the health-care system.[4-6]

PCGT 3.0 SETTINGS

Patient training settings include, but are not limited to:

- **3.1** Acute care hospital
  - **3.1.1** Patient's room
  - **3.1.2** Designated teaching area
  - **3.1.3** Pulmonary rehabilitation department
- **3.2** Outpatient rehabilitation center
- **3.3** Patient's home
- **3.4** Physician's office
- **3.5** Extended care or skilled nursing facility
- **3.6** Patient support group meetings[7]
- **3.7** Community education seminars (eg, industry- and school-based programs)[7]

PCGT 4.0 INDICATIONS

The presence of a patient population with the need to

4.1 increase knowledge and understanding of health status and therapy;[3]

4.2 improve skills necessary for safe and effective health care (ie, inability to perform needed therapy);[3]

4.3 foster a positive attitude, stronger motivation, and increased compliance with therapeutic modalities.[3,6,8]

PCGT 5.0 CONTRAINDICATIONS

There are no contraindications to patient and caregiver training when a need exists.

PCGT 6.0 HAZARDS/COMPLICATIONS

Omission of essential steps in care, inconsistency in information presented, or failure to validate the learning process can lead to untoward results.

PCGT 7.0 LIMITATIONS OF METHOD

7.1 Patient limitations:

7.1.1 Lack of motivation or interest in acquiring knowledge or skills. (This may include denial.)[7,9-12]

7.1.2 Impairment (eg, in hearing or vision, decreased energy or stamina, age-specific, pain, or medication side effects.)[2,7,8,10,13]

7.1.3 Inability to comprehend due to factors such as anxiety, depression, hypoxemia, substance abuse[8,14,15]

7.1.4 Negative response to past educational experiences or encounters[7,12,16]

7.1.5 Illiteracy despite level of education completed.[17,18] This may include functional illiteracy dealing with the healthcare process.[19]

7.1.6 A mind-set that leads to misapplication, misinterpretation, or rejection of instruction as irrelevant[7,12,16,20]

7.1.7 Language barriers[2,8]

7.1.8 Conflict of religious beliefs and/or cultural practices with material presented[2]

7.2 HCP limitations

7.2.1 Lack of positive attitude or adaptability[2,8,9,10,21]

7.2.2 Limited understanding of knowledge or skill to be taught[1,3,21,22]

7.2.3 Inadequate assessment of patient's need or readiness to learn and inability to individualize the instructional approach to the patient, including age-specific needs[2,8,20,21,23]

7.2.4 Multiple patient needs to be met in the allotted time[11]

7.2.5 Inappropriate or inadequate communication skills (eg, unnecessary use of medical terminology; lack of listening skills); lack of documentation or discussion with other team members; inconsistency in information presented[1,9,11]

7.2.6 Inadequate knowledge of cultural or religious practice that may affect educational process[2]

7.3 System limitations

7.3.1 Hospital stay too brief[7]

7.3.2 Absence of interdisciplinary cooperation[2] and communication

7.3.3 Inconsistency in information provided

7.3.4 Failure to provide interpreters[2,8]

7.4 Social limitations

7.4.1 Absence of support system[10,12,24]

7.4.2 Reimbursement issues[9-12]

7.5 Environmental limitations

7.5.1 Inadequate lighting, poor temperature control, uncomfortable seating, inadequate space for demonstrations[8,10]

7.5.2 Interruptions, distractions, and noise[8,10]

7.6 Poorly chosen resources, including inappropriate reading level and vocabulary[8,14,17,25]

PCGT 8.0 ASSESSMENT OF NEED

8.1 Determine the gap between what patient already knows and what he or she needs to know[4,7,8,26]

8.1.1 Interview patient regarding past experience with topic being taught.[7,15,16] A published or self-generated guide may be used.[13]

8.1.2 Discuss what patient perceives as knowledge relevant to his care. (A written test may be used to determine knowledge deficit in the context of pulmonary rehabilitation services. In many other clinical settings, a written test is not recommended)[15,16,20,21,26]

8.2 Observe patient's performance of therapy and determine whether skills are adequate for self-care. (If psychomotor skills are markedly impaired, an occupational or physical therapy assessment may be indicated.)[9]

8.3 Determine whether patient's attitude and outlook appear to be conducive to participation in his or her health care.

8.3.1 By observation and questioning, determine whether patient perceives himself as able to cope with his health care.[12]

8.3.2 By questioning patient and/or family, determine whether denial persists. (A psychological consult may be helpful.)[9]

8.3.3 Assess patient's motivation or emotional readiness to learn and change behavior as it relates to his health care.[2,6,8,9,12,15,21]

8.3.4 Consider using quality-of-life profiles to determine patient's general outlook and attitude or to determine the presence of low self-efficacy.[12,17,27]

PCGT 9.0 ASSESSMENT OF OUTCOME

9.1 Assessment of knowledge gained

9.1.1 In a nonthreatening manner, question patient about specific aspects of information presented and note questions asked.[10,16]

9.1.2 Request patient to repeat information given, in his own words.[16]

9.1.3 Paper and pencil tests may be used but are not the recommended approach.[15,16]

9.1.4 Note behavior changes that appear to be manifestations of increased knowledge.

9.2 Assessment of skills mastered

9.2.1 Observe patient performing skill, without prompting or assistance.[2,10,28]

9.2.2 Discuss the new skill with patient and question him as appropriate.

9.2.3 Observe patient's ability to adapt new skill to novel or unfamiliar situations.[13]

9.2.4 Determine skill adaptability in home environment by phone calls and home visits.[10]

9.3 Assessment of patient outlook and attitude

9.3.1 Observe patient and quantitate appropriate variables for evidence of life style changes (eg, weight change, smoking cessation, increased physical activity).

9.3.2 Determine through discussion with patient and family whether patient feels more in control of condition and care.[9]

9.3.3 Use quality of life inventories; such as the COPD Self-Efficacy Scale,[29] the Chronic Respiratory Disease Questionnaire,[30] the SF-36,[31] the Sickness Impact Profile,[32] or CES-D (Center for Epidemiologic Studies Depression Scale).[33]

PCGT 10.0 RESOURCES

10.1 Essentials of the training process (See the Appendix).

10.2 Training materials

10.2.1 Written material

10.2.1.1 Readability and comprehension should be at approximately the 5th-6th grade level. (This may be assessed by the SMOG scale.)[14,17,20,25] A glossary may be helpful.

10.2.1.2 Large, dark print on white page is preferable.[15,18]

10.2.1.3 Material should be easy to handle with space for instructions and/or notes.[8]

10.2.1.4 Simple terms and short sentences encourage use of material.[8,17]

10.2.1.5 Illustrations may aid comprehension.[18,34]

10.2.2 Audiovisual equipment (eg, videocassette player, audiotapes, slides, models) may be helpful.

10.2.3 Computers and software may be useful both for instruction and documentation.[7,23,28]

10.3 Training site should be an appropriate environment with adequate space, few interruptions or other distracters, adequate lighting and temperature, and comfortable seating.[2,8,10]

10.4 Personnel: All HCPs should have patient training skills commensurate with the level of care that they provide to the patient.[2] Skills should include but are not limited to

10.4.1 appropriate communication skills, including verbal, nonverbal, and listening skills;[9,14,16,20,21]

10.4.2 compassion, empathy, and a nonjudgmental attitude;

10.4.3 good knowledge of principle and theory, with the ability to present information in a manner consistent with that of the rest of the health-care team;[9,21,22]

10.4.4 ability to use appropriate language levels in written and verbal instruction;[1,10,20,25]

10.4.5 ability to be patient, use repetition, project a positive attitude, and build rapport;[1,8,10,12,15,20,21]

10.4.6 ability to view patient as individual and to assess all aspects of his educational needs and limitations, including those of an age-specific, psychosocial, and cultural nature.[2,7,9,15,20,21]

PCGT 11.0 MONITORING

11.1 Monitoring the patient training process should include awareness of patient's verbal and nonverbal responses to what is being taught, noting

11.1.1 good eye contact vs drowsiness,
11.1.2 active listening vs preoccupation,
11.1.3 active discussion vs silence.

11.2 Observation and questioning allow for ongoing patient monitoring.

11.3 The assessment of readiness for self-care may be made by working with the discharge planner.

11.4 Patient's instruction and the outcome should be documented (See Appendix).

PCGT 12.0 FREQUENCY

12.1 Spontaneous—as often as patient contact is made.

12.2 Schedule patient appointments as needed.

12.3 Scheduled group sessions may meet as often as once or twice a week up to once a month.

12.4 Closed-circuit hospital video presentations may be presented several times on a given day and repeated as indicated.

PCGT 13.0 INFECTION CONTROL

13.1 Implement Universal Precautions and tuberculosis control measures as indicated.[35,36]

13.2 Observe all infection control guidelines posted for patient.

13.3 Any equipment used should be appropriately cleaned and stored.

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APPENDIX

(References are listed following the body of the Guideline)

Essentials of the Training Process

Health-care professionals should know how to teach and how to modify their teaching approach to provide the desired outcome.

I. Complete a needs assessment to establish measurable goals and outcomes for patient training.

A. Involve the patient or family as active participant(s) in goal setting. (Family refers to the person or persons who play a significant role in the patient's life. This may include individuals who are not legally related to the patient.)[2,16]

B. Set realistic goals for the individual patient.[8,10]

II. Try to determine how the patient learns best through discussion of past educational experiences, observation, or experimentation with different teaching tools:[2,9,16]

A. Visualization, or learning by reading;

B. Auditory, or learning by listening;

C. Tactile/psychomotor, or learning by doing.

III. Select the training approach and style that you believe will most benefit the patient. This may vary as the patient situation changes. No one approach is inherently superior to another.[2,7,12,20,27]

A. One to one and/or group sessions.

B. As a rule, use simple vocabulary and short phrases tailored to the patient's life experiences.[4]

C. Encourage active patient participation in the training experience (often more effective than didactic teaching).[1,7,12,16]

D. Speak slowly and clearly, lower your voice, and stand where you can easily be seen.[10,15]

IV. Present material in organized sequence using appropriate terminology.[1,20]

A. Ensure the patient's attention.[6,26]

B. Discuss the learning objectives to be addressed and their relevance.[1,20,26]

C. Review what the patient may know from past experience, and build on similarities and differences compared to current topic.[12,20,26,28]

D. Present specific information in a concise, clear manner using short words and sentences.[6] Progress from the simple to the more complex.[20]

E. Ask the patient to determine and articulate the concept or technique taught.[26]

F. Patiently repeat important points, answer questions, and give specific feedback for correction of technique theory.[1,15,20,26]

G. Reinforce objectives through positive feedback.[12,20,26,28]

V. Document what has been taught (link to expected learning outcome) and at what level the patient has mastered knowledge and skill and has improved behavior. Incorporate this information into the medical record in a clear, concise manner that communicates needed information to other team members.[2,7,23,28]

VI. For patient training to be effective, on-going reinforcement and follow-up are needed.

A. Provide written material as review.[3]

B. Activate support system.[3,6,23,24]

C. Allow time for repeated patient demonstration of learned material; incorporate a behavioral and skills mastery emphasis.[1,6,20,28]

D. If appropriate, allow for periodic follow-up phone calls.[10,13,23]

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An Atlas of Lung Infections, The Encyclopedia of Visual Medicine Series, by JF Costello, DE Saunders, and J Philpott-Howard, with a Foreword by PC Hopewell. Hardcover, 112 pages, 141 illustrations (67 color illustrations), index, and bibliography. London: Parthenon Publishing Group, 1996. $85.

Respiratory infections are an important cause of morbidity and mortality in children and adults. This is a worthwhile topic for The Encyclopedia of Visual Medicine, a series that intends to provide a visual approach to clinical medicine. The authors, who are staff members from King's College School of Medicine and Dentistry and St George's Hospital Medical School in London, attempt to combine clinical, radiologic, and microbiologic approaches to common respiratory infections. Physicians-in-training are the intended audience, yet senior physicians, nurses, and respiratory care practitioners may find this book of interest.

This slim volume is printed on large (30 × 20 cm or 11 5/8 × 8 in.) pages of heavy, coated paper. It consists of a short listing of contents, a foreword, a brief introduction to the clinical and diagnostic approach, and vignettes about bronchial infections, community-acquired pneumonias, hospital-acquired pneumonias, tuberculosis, and infections associated with the acquired immune deficiency syndrome (AIDS). Section 2 (74 pages) contains the illustrations. Section 3 is a bibliography of 35 references; absent are helpful comments that direct the reader to the correct reference.

Section 2 is the raison d'être of this atlas. About half of the illustrations are radiologic studies consisting of plain chest radiographs, a few computerized tomographs, a bronchogram, and a magnetic resonance image. The rest are either photographs of pathogens identified by clinical laboratory tests (eg, traditional microbiology methods or cultures, tissue culture, wet-mount microscopy, tissue histology, electron microscopy) or are photographs of the special tests used to establish the diagnosis (eg, counter-current immunoelectrophoresis, complement fixation, hemagglutination inhibition, latex agglutination, and enzyme-linked immunosorbent assay).

Sadly, there is only a *single* patient image—finger clubbing. This violates the authors' stated objective of "providing a guide to the presentation and diagnosis of infections." In contrast, there are 2 images of blood culture bottles (Figs. 24 and 129), a nasopharyngeal swab (Fig. 49) and a color strip test for β-lactamase (Fig. 6)—frivolous given the high cost of publishing color images. I was dismayed at the amount of unused space—roughly 30% of each page. Some pages are white below the captions as in Figures 1 and 2, and some include a gray mat that surrounds each image rather than good, accurate descriptions of the image.

In addition, some images are disappointing. I was appalled by the poor focus and lack of contrast of the radiographs for chronic obstructive pulmonary disease (Figs. 1 and 2). Other images are simply too small to demonstrate the findings described in the legends and should have had accompanying detailed enlargements such as ring shadows and band shadows in cystic fibrosis (Fig. 13), pneumatoceles in staphylococcal pneumonia (Fig. 27), small nodules in varicella pneumonia (Fig. 54), and miliary shadowing in tuberculosis (Fig. 112). Others fail to capture subtle phenomena; for example, the latex agglutination test for cryptococcal antigen appears as 2 red circles on a black field (Fig. 133).

Some legends try to explain too much in the allotted space as in these examples: complement fixation test for mycoplasma (Fig. 35), the hemagglutination inhibition test for influenza antibodies (Fig. 45), and the rapid micro-agglutination test for legionella (Fig. 73). Another example, immunofluorescent antibody test for legionella (Fig. 73), contains distracting information about additional tests that are not shown. The caption for the hydatid cyst is confusing (Fig. 97); the large lobulated cyst is of interest despite being an incidental finding. Finally, some captions are inaccurate, for instance, agglutination of erythrocytes (clumping and rouleaux formation) is shown, not cold agglutinins (Fig. 34); colonies of aspergillus mold appear to be growing on blood agar (or brain-heart infusion medium), not Sabouraud's agar (Fig. 86).

However, some images will be instructive for clinicians who have forgotten about the mucoid strain of *Pseudomonas aeruginosa* (Fig. 17), the alpha-hemolysis and optochin sensitivity of *Streptococcus pneumoniae* (Fig. 21), and the coagulase reaction of *Staphylococcus aureus* (Fig. 16). The images of the immunofluorescent stain for respiratory syncytial virus (Fig. 44), the black colonies of *Bacteroides melaninogenicus* (Fig. 95), the swarming of *Proteus mirabilis* on agar (Fig. 102), the fluorescent auramine stain for mycobacteria (Fig. 116), and the gold colonies of *Mycobacterium kansasii* (Fig. 131) are memorable.

A fascination with tradition may explain the continued separation of the clinical (primarily textual) and laboratory (primarily visual) approaches, the dearth of patient images, the paucity of detailed radiographs, the frivolous images, the wasted space, and the inability to determine what the clinician needs to know. Inadequate technical resources may explain the poor illustration quality, composition, and lay-out. However, journals, such as the *British Medical Journal* (ABC Series), and textbooks, such as the *Atlas of Pediatric Physical Diagnosis* (Second edition. Zitelli BJ, Davis HW, editors. London: Mosby-Wolff, 1992) and the *Atlas of Bronchoscopy* (Becker HD, Kayser K, et al, editors. Philadelphia: BC Decker, 1990) have overcome these obstacles and provide well-integrated textual and visual information, side-by-side on the same page or on facing pages.

As I reviewed the **Atlas**, I wondered why the text in the clinical vignettes (Section 1, Pages 17-32) was not placed in the empty white and gray spaces adjacent to each image (Section 2, Pages 35-104) along with patient images, detailed images, and helpful clinical comments. I also wondered why more text was not devoted to explaining the efficacy of the test, the duration of illness required before the test may be helpful, the turn-around time, and when to obtain confirmatory tests.

Schering Corporation distributed a helpful book, *Diagnostic Aid to Respiratory Infection* (JL Le Frock, 1979) as did Abbott

Laboratories, *Schneierson's Atlas of Diagnostic Microbiology* (EJ Bottone, 1982)—both free of charge to the reader. Some of the information is dated—before AIDS and the hantavirus were discovered—so it will be necessary to consult current texts such as Koneman's *Introduction to Diagnostic Microbiology* (Philadelphia: JB Lippincott, 1994) and Pennington's *Respiratory Infections: Diagnosis and Management, Second Edition* (New York: Raven, 1989). If you cannot find these gems or if looking for updated information is too bothersome, then you might try the **Atlas of Lung Infections**.

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100 Statistical Tests, by Gopal K Kanji. Softcover, illustrations, and tables of distributions, 216 pages. London, Thousand Oaks, CA, & New Delhi: Sage Publications Inc, 1994. $22.95.

The title of this book intrigued me, and so I began—there really are 100 statistical tests, and more, according to the author. The stated goal for this text is "to provide a reference book for both the applied statistician and everyday user of statistics. ... Lucidity of style and simplicity of expression have been [our] twin objectives...", concludes the preface.

Some ambiguity can be found among texts of statistics in terms and symbols, and Dr Kanji defines those he prefers and uses them consistently. The introduction is brief and somewhat fragmented probably because it is designed only to explain the concepts required to understand the format/template and terms used to describe the 100 tests. The template is the same for each test:

- Statement of the null and all forms of the alternative (or action) hypotheses
- Formula for the test statistic
- Setting in which the test is used
- Critical region of the distribution
- Data (n, h, *s*, and others)
- Conclusions under both the null and alternative hypotheses
- Illustrations as applicable

The next section provides several examples and exercises in using this template (Fig. 1). Then come the 100 tests—from the familiar Z Test (Test 1), to the less-familiar but useful Dixon Test for Outliers (Test 22), and concluding with one that is new to me—analysis of variance for angular data (Test 100). A list and brief description of the tests in the order they are covered and by category are provided.

A 26-reference bibliography and a topic index follow 39 tables. I am disappointed that the tables do not include instructions for their use.

Although the author states "an elementary knowledge of statistics should be sufficient to allow the reader to carry out the test," in my opinion, **100 Statistical Tests** is not a book for the statistically uninitiated but may be a useful reference. As books on statistical analysis go, it is a real value.

Kaye Weber MS RRT
Associate Editor
RESPIRATORY CARE
Dallas, Texas

Test 1 *Z*-test for a population mean (variance known)

Hypotheses and alternatives

1. H_0: $\mu = \mu_0$
   H_1: $\mu \neq \mu_0$
2. H_0: $\mu = \mu_0$
   H_1: $\mu > \mu_0$

Test statistics

$$Z = \frac{\bar{x} - \mu_0}{\sigma/\sqrt{n}}$$

n is sample size
$\bar{x}$ is sample mean
σ is population standard deviation

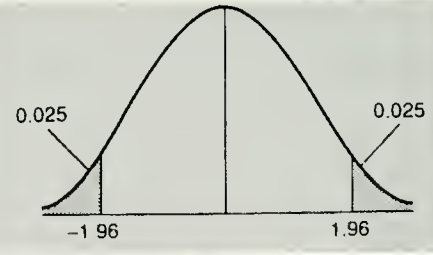
When used

When the population variance σ^2 is known and the population distribution is normal.

Critical region and degrees of freedom

Using $\alpha = 0.05$ [see Table 1]

1.



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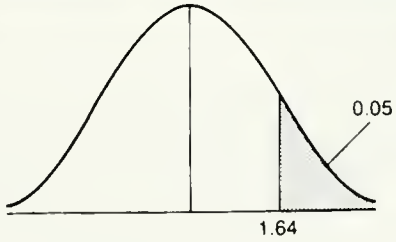

Data

H_0: $\mu_0 = 4.0$
$n = 9$, $\bar{x} = 4.6$
$\sigma = 1.0$
$\therefore Z = 1.8$

Conclusion

1. Do not reject H_0. [see Table 1]
2. Reject H_0.

Books, references, and directories now available from the publishers.

Allied Health and Rehabilitation Professions Education Directory 1996-1997, 24th Edition, by the American Medical Association. Softcover, 400 pages, includes education accreditation agencies, occupational descriptions, institutions where studies are available, and allied health and rehabilitation education data. Chicago: American Medical Association, 1996. $44.95 for AMA members; $54.95 for nonmembers. Call (800) 621-8335 to order.

Physicians Desk Reference® Generics™ by Medical Economics, 2nd Edition. Hardcover, 3,100 pages, includes cost of therapy tables, international drug name index, generic drug monographs, major brand products and FDA bioequivalent alternatives, visual identification guide, imprint identification guide, and 3 quick-reference indices. Montvale NJ: Medical Economics Company, 1996. $79.95. For more information or to order, call (800) 232-7379.

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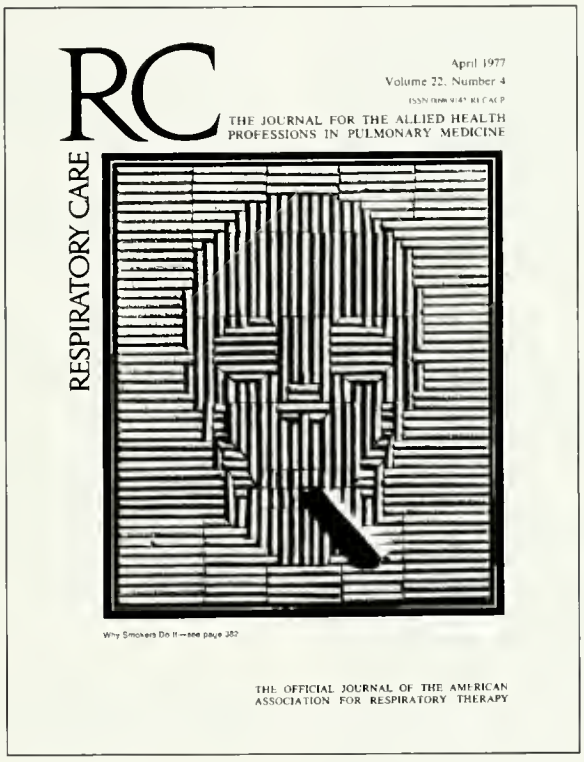
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RESPIRATORY CARE
April 1977
Volume 22, Number 4
THE JOURNAL FOR THE ALLIED HEALTH PROFESSIONS IN PULMONARY MEDICINE
Why Smokers Do It—see page 382
THE OFFICIAL JOURNAL OF THE AMERICAN ASSOCIATION FOR RESPIRATORY THERAPY

Why Smokers Do It

Whatever we've heard till now about the urge to smoke may or may not be true; psychological explanations valid for one smoker rarely apply to all smokers.

Columbia University Psychologist Stanley Schachter has proposed an answer that fits all smokers: nicotine intake reduces acidity in the urine, which usually rises as a direct result of stress on the system. In a brief report in *Time* (Feb. 21, 1977) and a full-length article in the March 1977 issue of *Journal of Experimental Psychology*, Schachter says, "the smoker's mind is in the bladder. ... When plasma nicotine is below the smoker's usual level, he smokes; when it is at his level, he doesn't."

If you're a smoker, what can you do in stressful situations to avoid increasing your intake of nicotine? Schachter suggests taking bicarbonate of soda. The "plop-plop, fizz-fizz, oh what a relief it is," of Alka-Seltzer commercials could be as effective before a party as after. Schachter and others also recommend lobbying for high-nicotine, low-tar, low-gas cigarettes.

We have a better suggestion: stop.

The AART Zero-Smoking Committee is currently gathering information about smoking-cessation techniques to help AART's smoking members to kick the habit. When the committee has finished the project, says chairman John Beree, AART, of Doctors Medical Center Hospital, Tulsa, Oklahoma, smokers may be able to participate in AART- or affiliate-sponsored smokers' clinics or be referred to other group therapy sessions in their areas. Information or experience you may have with any of these techniques would help the committee to develop this service so much faster.

Until that happens, we suggest all you who are smokers try to stay calm. *[Respir Care 1977;22(4):382]*

RESPIRATORY CARE

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Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References, Tables, Appendices, and Figures and Captions may be included.

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Blood Gas Corner: A brief, instructive case report involving blood-gas values—with Questions, Answers, and Discussion.

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Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

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Cardiorespiratory Interactions. A case report demonstrating the interaction between the cardiovascular and respiratory systems. It should be a patient-care scenario; however, the case—the central theme—is the systems interaction. CRI is characterized by figures, equations, and a glossary. See the March 1996 issue of RESPIRATORY CARE for more detail.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs, may involve imaging techniques other than conventional chest radiography.

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Article in a journal carrying pagination throughout volume:

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Article in a publication that numbers each issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

6. Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

7. Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

8. Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

10. DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura, 1990:76-85.

Corporate author book:

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Chapter in book with editor(s):

12. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

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British Medical Journal 1995;311:1272 and Noted by JFL MD in Pediatrics 1996;97(4):A24.

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- ☐ 6-10 years
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Coming in the August Issue of RESPIRATORY CARE!

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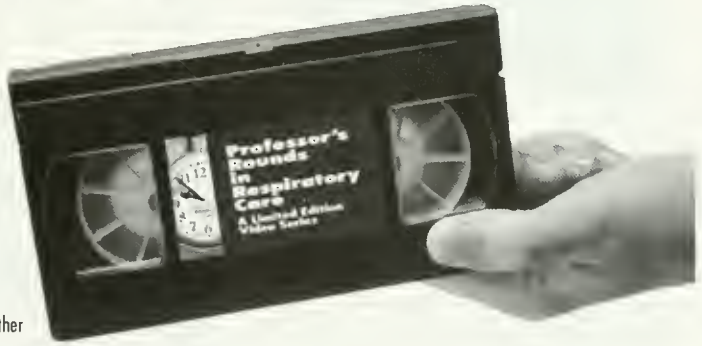
Only $5

Here's one you've really got to see!

The New JCAHO Hospital Standards: Their Effect on Respiratory Care

A Videotape from the Professor's Raunds in Respiratory Care Videoconference Series

Featuring Nancy Telford, BS, RRT, Program Monoger, Joint Commission on Accreditation of Healthcare Organizations, with Moderator Sam P. Giardano, MBA, RRT, AARC Executive Director



The new *1995 Accreditation Manual for Hospitals* is now organized around functions that impact patient outcomes, and the standards are now integrated into functional chapters rather than a single chapter. Through this overview, you will learn about standards related to competence programs, responsibilities of department leaders and medical directors, and expectations on performance improvement. This video also reviews the interactive survey process and the role of department staff. **90 minutes. Item VC51 — $275** ($305 nonmembers)

Please send me "The New JCAHO Hospital Standards: Their Effect on Respiratory Care." Item VC51 — $275 ($305 nonmembers)

Payment enclosed $_____ Charge to Purchase Order #_____ *Please Note: Videotapes do not qualify viewer for CRCE credit.*

Charge to my ☐ Visa ☐ MasterCard. Card expires ___/___ Card #_____ Signature_____

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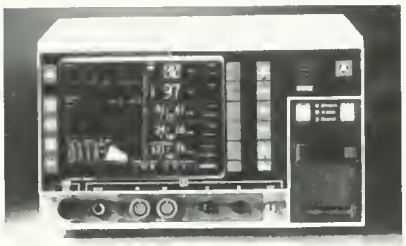
New Products & Services

News releases about new products and services will be considered for publication in this section. There is no charge for these listings. Send descriptive release and glossy black and white photographs to RESPIRATORY CARE Journal, New Products and Services Dept, 11030 Ables Lane, Dallas TX 75229-4593.

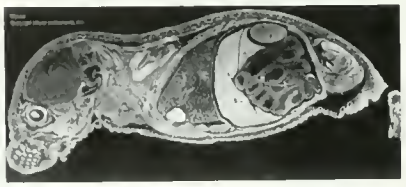


VITAL SIGNS MONITOR. BCI International adds the Vital Signs Monitor, Model 6200, to its current product line. The unit consists of noninvasive blood pressure, pulse oximetry, and electrocardiographic monitoring. Patient information is displayed on a high resolution screen. Options for the monitor include printing functions, 2 channels of pressure and/or 2 temperatures, dedicated hard keys, or menu-driven software. Waveforms are configurable and can be tailored to meet a broad range of clinical needs. To query the company, circle reader service card number 90.

ASTHMA HOME CARE PLAN. Raiinbow Step Up Systems Incorporated releases kits for the home treatment of asthma. Recently approved by the FDA, the Raiinbow™ Asthma Home Care Plan is designed to correspond with the color-coded guidelines issued by the National Heart, Lung, and Blood Institute. According to Raiinbow, the green kit for mild asthma includes a peak flow meter; the yellow kit for moderate asthma includes a peak flow meter, a nebulizer, and all necessary accessories; and the red kit for severe asthma includes 2 peak flow meters, pulse oximeter, oxygen tank, nebulizer, and all necessary accessories. The kits are available by prescription only and packaged in corresponding colors. For a complete guide to these kits, circle Raiinbow's reader service card number 91.

Infant Heel Warmer. Florida Medical Industries Inc announces the availability of the Recover® Infant Heel Warmer. The warmer has a self-adhesive strap that enhances atraumatic vasodilation and increased blood flow in an infant's heel to ensure adequate blood for sampling, according to the company. The warmer is made from safe, nontoxic, food-grade sodium acetate, with an easy-to-activate catalytic disk. The warmer can maintain a consistent temperature of 105° F. Circle reader service card number 92 for more details.

Ultrasonic Nebulizer. DeVilbiss releases a new design for the Pulmosonic™ Ultrasonic Nebulizer. The Nebulizer is a good choice for patients who find metered dose inhalers difficult to use, the company claims. The PulmoSonic operates from household current, a car lighter adapter, or an optional battery pack. The nebulizer features a patient-demand system that allows the patient to control the delivery rate of the medication. The system also works well with a mask. The optional accessory kit includes a carrying case, rechargeable battery and instructional video. For more information, circle reader service card number 93.



Specimen Scanner. Polaroid Electronic Imaging Systems and Meyer Instruments Inc have created a new whole-mount histologic or biologic image scanner, the SprintScan 35 and PathScan Enabler. According to the company, researchers and pathologists among others can use the process to capture images of whole specimens at a high resolution. The PathScan Enabler is an aluminum holder with a proprietary, optically flat neutral-density filter, developed by Meyer Instruments. A 36 × 24 mm glass specimen slide can be inserted into the holder, that can be inserted into the Polaroid SprintScan 35 scanner that digitizes the images up to 2,700 dots/in. In < 1 minute, the scanner produces a digital image that can be edited, measured, annotated, and printed. The scanner is available for both Macintosh and Windows platforms. For details, circle reader service card number 94.

SURGICAL GLOVES. American Pro Latex releases the GANTEX line of sterile surgeon gloves. American claims that the gloves are thicker and stronger, offering more protection, fewer pin holes, and reduced risk of allergic reaction. For information about the gloves, circle reader service card number 95.

MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Page of

Form Approved: OMB No. 0910-0291 Expires: 4/30/96
See OMB statement on reverse

FDA Use Only (Resp Care)

Triage unit sequence #

A. Patient information

1 Patient identifier
In confidence

2 Age at time of event:
or
Date of birth:

3. Sex
☐ female
☐ male

4 Weight
____ lbs
or
____ kgs

B. Adverse event or product problem

1 ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2 Outcomes attributed to adverse event (check all that apply)
☐ death (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other:

3 Date of event (mo/day/yr)

4 Date of this report (mo/day/yr)

5 Describe event or problem

6. Relevant tests/laboratory data, including dates

7 Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1 Name (give labeled strength & mfr/labeler, if known)
#1
#2

2 Dose, frequency & route used
#1
#2

3 Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5 Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8 Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9 NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1 Brand name

2. Type of device

3 Manufacturer name & address

4 Operator of device
☐ health professional
☐ lay user/patient
☐ other:

5. Expiration date (mo/day/yr)

6
model #
catalog #
serial #
lot #
other #

7 If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9 Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1 Name & address

phone #

2. Health professional?
☐ yes ☐ no

3 Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5 If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 1/96)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 to report by phone or for more information
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity, including the identity of a self-reporter, may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

The public reporting burden for this collection of information has been estimated to average 30 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to:

DHHS Reports Clearance Office
Paperwork Reduction Project (0910-0291)
Hubert H. Humphrey Building, Room 531-H
200 Independence Avenue, S.W.
Washington, DC 20201

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Please do NOT return this form to either of these addresses.

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Public Health Service • Food and Drug Administration

FDA Form 3500-back

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The FDA Medical Products Reporting Program
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5600 Fishers Lane
Rockville, MD 20852-9787

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Calendar of Events

AARC & AFFILIATES

August 27—AARC Video Conference
"Theory and Application of Neonatal Ventilation: What, When, and Why," Part 5 of the 1996, 7-part video conference "Professor's Rounds in Respiratory Care," from 12:30 to 2 p.m. Eastern Time. Live telephone question-and-answer session airs Sept. 16. CRCE: 1 credit hour. Contact: AARC at (214) 243-2272.

September 11–13—Maryland/ District of Columbia Society
15th Annual Conference by the Sea at the Carousel Hotel in Ocean City, Maryland. Contact: Jacqueline Smith at (703) 719-3916 or Elgloria Harrison at (202) 547-6817.

October 2–4—Kentucky Society
Annual state meeting, "Strive for Excellence," at the Hurstborne Conference Center in Louisville, Kentucky. Contact: Janet Vogt at (502) 562-3002.

OTHER MEETINGS

September 26–27—INFOCARE
First Annual Neonatal/Pediatric Respiratory/Critical Care Symposium at the Sheraton Beach Resort in South Padre Island, Texas. Contact: INFOCARE, 350 San Roman Rd, Los Fresnos, TX 78566, (210) 233-5645.

November 10–14—Asia Pacific Association for Respiratory Care
5th Congress of APARC at the Bangkok Convention Centre and Central Plaza Hotel in Bangkok, Thailand. Abstracts must be submitted by July 31. Contact: Congress Secretariat, 5th Congress of APARC, Congress and Convention Experts (Thailand) Co Ltd, PO Box 10-163, Ladphrao, Bangkapi, Bangkok 10310, THAILAND.

March 20–22, 1997—American Lung Association of Montana
16th Annual Big Sky Pulmonary & Critical Care Medicine Conference at the Big Sky Ski & Summer Resort in Big Sky, Montana. Contact: ALA of the Northern Rockies, 825 Helena Ave., Helena, MT 59601-3459, (406) 442-6556, fax (406) 442-2346.

November 3-6, 1996, Sunday-Wednesday

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3. ❑ Hosp 200 to 299 beds
4. ❑ Hosp 100 to 199 beds
5. ❑ Hosp 100 or less beds
6. ❑ Skilled Nursing Facility
7. ❑ Home Care Practice
8. ❑ School

II. Department
A. ❑ Respiratory Therapy
B. ❑ Cardiopulmonary
C. ❑ Anesthesia Service
D. ❑ Emergency Dept

III. Specialty
1. ❑ Clinical Practice
2. ❑ Perinatal Pediatrics
3. ❑ Critical Care
4. ❑ Clinical Research
5. ❑ Pulmonary Function Lab
6. ❑ Home Care/Rehab
7. ❑ Education
8. ❑ Management

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C. ❑ Supervisor
D. ❑ Staff Technician
E. ❑ Staff Therapist
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8. ❑ Management

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C. ❑ Supervisor
D. ❑ Staff Technician
E. ❑ Staff Therapist
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G. ❑ Medical Director
H. ❑ Anesthesiologist
I. ❑ Pulmonologist
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REFERENCES 1. Data on file. HealthScan Products Inc. 2. Wheeler BB, Bollers ND. Initial experience with a novel spacer device. (Presented at the 1993 meeting of the American Academy of Allergy and Immunology) * U.S. Patent No. 5,040,527 OA760004-0 © 1994, HealthScan Products Inc

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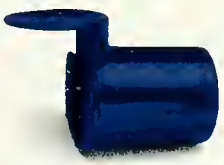
And because more convenience usually means better compliance, OptiHaler packs all of these advantages into a sleek, discreet, truly portable unit that patients rate significantly more convenient for daily use than the leading holding chambers.[2]



New, more convenient, one-piece cap.

So why not make MDI therapy easier for your patients...with the product more patients prefer.[2] Call our 24-hour customer service hotline today for your free professional sample of new, improved OptiHaler.

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NEW IMPROVED

OptiHaler®

Drug Delivery System for use with Metered Dose Inhalers

The promise of MDI therapy...delivered.

From the makers of ASSESS® and Personal Best® Peak Flow Meters

HealthScan Products Inc., Cedar Grove, NJ

REFERENCES 1. Data on file. HealthScan Products Inc. 2. Wheeler BB, Bolfers ND. Initial experience with a novel spacer device. (Presented at the 1993 meeting of the American Academy of Allergy and Immunology) * U.S. Patent No. 5,040,527 OA760004-0 © 1994, HealthScan Products Inc.

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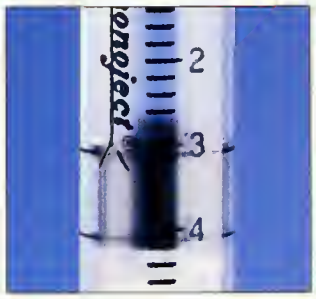
INTRODUCING

3 NEW Technologies in Arterial Blood Sampling

ASPIR-PULSE™ Arterial Blood Gas System

NEW, Advanced ASPIR-PULSE™ Syringe

Patented design improves filling for both **Aspir**ation and ***Puls***ation techniques



NEW, Purge Guard™
One-Handed Safety Needle Venting System

Patent-pending design allows One-Handed operation to immediately **Purge** Air Bubbles, immediately **Guard** the needle point and immediately free the other hand to apply pressure at the puncture site

NEW, Total Ca^{++} Lyte™
Precision Heparin

A breakthrough patented heparin to maximize the precision of test results obtained from the new critical-care blood gas and critical analyte analyzers

	Non-Heparinized Control	Aspir-Pulse Total Ca^{++} Lyte
pH	1.22[1] 1.24[2]	1.23[1]
pCO_2	2.38[1]	2.35[1]
pO_2	0	12[1]

See your Sherwood Medical O.R./Critical Care Sales Representative or call 1-800-325-7472 for a complete listing of ***ASPIR-Pulse***™ Arterial Blood Gas Kits.

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